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THE END-RESULTS OF SEVENTY CASES OF RENAL TUBERCULOSIS TREATED BY NEPHRECTOMY [1]

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INTRODUCTION

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THOUGH many points connected with the diagnosis and treatment of renal tuberculosis have been practically settled during the past ten years, much still remains to be done. In expert hands the questions of diagnosis have been well cleared up and the formerly much discussed question of the advisability of surgical treatment has ceased to play an important part. The tendency to watchful waiting which was generally but another name for medical procrastination has considerably abated and there is practically no difference of opinion, among those qualified to express one, that in unilateral renal tuberculosis operation offers the only chance of cure and the so-called medical treatment only prolongs the agony.

The scientific world is still waiting for proved cases of renal tuberculosis healed permanently, except by removal of the kidney, either by nature or by art. But though in the field of diagnosis and in the principles of treatment general agreement has been obtained, our knowledge of what actually happens to these patients following nephrectomy is often largely a matter of speculation. Far too much we have depended upon our impressions, which are notably inaccurate, and have assumed that from these patients no news was good news.

It is, therefore, with great pleasure and much profit that I have followed my associate, Dr. Crabtree, in his study of the results of nephrectomy for renal tuberculosis at the Massachusetts General Hospital. The work has been carried out with a care as to detail which will satisfy the most captious and also with complete willingness to accept the showing, no matter what it may be.

His paper raises in my mind again certain questions which have troubled me in the past and upon which it seems to me further work is necessary.

In every series of reported cases of considerable size, as in most of the other operations dealing with tuberculous lesions, there is a certain proportion of the patients who die promptly from general miliary tuberculosis. In addition to this there is a considerable number of patients who, following operation, have a high temperature, a rapid pulse, and all the evidences of profound toxæmia. Most of these patients recover. The minority die with miliary tuberculosis.

[1] From the Records of the Massachusetts General Hospital.

I have been on the whole surprised that this phenomenon has not been more frequent, as, in our efforts to free these generally adherent kidneys, the probability of forcing a considerable amount of toxic material, and even actual bacilli, into the circulation seems overwhelming. I have been led to wonder whether this might not be avoided by an operation planned so as to secure the vessels before manipulation of the kidney. As our technique improves and our mortality becomes lower, this question must certainly be taken up and unless it should appear that the renal vein is not the chief source of infectious supply it would seem to me an important consideration.

The cases here reported by Dr. Crabtree, and particularly a number of recent cases of my own, have brought out the fact that operation upon early lesions is no more likely to be attended by a good ultimate result than is operation upon much more advanced lesions. This may, of course, depend upon at least two conditions: first, that the renal tuberculosis is only an evidence of a generally low resistance to the tubercle bacillus and that the failure of nephrectomy to control the process proved nothing except that it could not have been controlled by any method; or second that the process in the kidney is so early that there has developed no immunity and that if left to itself for a short time such heightened resistance might come. Certainly this apparent fact requires careful consideration and we must either modify our technique so as to avoid the possibility of spreading the infection or take measures to hasten the development of immunity.

While it would seem a pity to do anything which remotely tended to discourage the only just developing enthusiasm for removing the tuberculous kidney at the earliest possible moment, it is clearly necessary to call attention at the present time to what our experience has led us to believe to be a not inconsiderable danger; viz., the lack of resistance in patients with early tuberculous lesions of the kidney.

THE fate of the nephrectomized in renal tuberculosis is not yet a closed book. It is with the purpose of adding to the literature of this important subject that the following data are presented.

The cases studied comprise those treated since the establishment of nephrectomy as a routine treatment for renal tuberculosis, up to December, 1912. It was not considered advisable to include those cases which were operated upon since this date and which were of less than two years' standing after nephrectomy at the time of beginning this investigation, since this period is not sufficiently long to allow for the recovery of bladder symptoms in the average case. The nephrectomies were performed by a number of surgeons, represent many varieties of technique, and approximate the average results of good general surgical skill. The cases operated upon in the genito-urinary service in the first two years of its existence are also included. Of these, the great majority was operated upon by Dr. Hugh Cabot.

The records of the hospital contain 103 cases operated on within the period designated. Of these, 4 patients died in the hospital. These deaths will be discussed later. Letters were sent out to the 99 patients who left the hospital alive, requesting a visit to the hospital for examination. If the patient were still alive but unable to visit the hospital, he was requested to send a specimen of urine and data as to his condition.

Fifty-one cases reported and were examined or sent letters with urine; more than three-quarters, 45, were seen in person. Eleven letters from friends of patients or from the city clerk report the patient as dead. In the majority of these, the cause of death is given but no necropsies are available. The clinical diagnosis is given and in advanced tuberculosis it is usually a satisfactory explanation of the cause of death. In 19 instances the letters were not returned. In 8 of these cases the records contain notes made one to four years after operation. It is reasonable to suppose that not all of these are dead but

that some are lost through change of address. In 10 instances the letters were returned unclaimed and all efforts to trace the patients through friends or from records of death certificates were fruitless. In 70 of the 99 cases, definite data, more or less complete, were obtained.

The average duration of time since operation, for the series, exclusive of the 4 cases of immediate post-operative death, is 5.6 years. One patient is living 18 years after nephrectomy; one, 15; one, 13; two, 11; and one, 10 years.

The following data were obtained from the hospital records:

Sex. The patients were about equally divided as to sex, there being 34 males and 36 females. In the majority of series reported there is a slight preponderance of males. In 1,176 cases reported by von Frisch, Wildbolz, Asakura, Walker, Guilliard, Tuffier, Albarran, and Braasch there are 633 males and 543 females.

Wildbolz mentions that among women there are larger percentages of cures. This observation is borne out by the fact that in the 9 "unimproved" cases, 8 are male and 1 female. And of the late deaths, 11 are male and 3 female.

Age. There is 1 case of tuberculosis in childhood in this series, a boy 14 years of age. The average age for the series is 34.9 years with extremes of 14 and 65 years. It is well known that renal tuberculosis is a disease of young adult life and these statistics accord with this observation. Braasch found that only 29 per cent of his series were above 40 years of age. Wildbolz reports that of a series of 316 cases, 224 were between 20 and 40 years of age, while only 4 were under 10. Asakura found that of 70 cases, 60 were within the above limits, while none was under 10. Forty-one, or 58.5 per cent, of our cases occurred in the third and fourth decades. The ages as shown in decades are as follows:

	Cases
11 to 20 years	6
21 to 30 years	17
31 to 40 years	24
41 to 50 years	12
51 to 60 years	3
61 to 70 years	1
Age not given	7

A striking fact to be observed is that in the 4 cases in which the patients were over 50 years of age, the lesions were calcareous kidneys with autonephrectomies.

Side. No cases of bilateral renal tuberculosis were knowingly operated upon. It is probable that were these cases seen under the conditions of our present-day clinics and with present-day methods of diagnosis applied, some would be demonstrated to be bilateral infections. A review of our recent out-patient cases shows a larger number of bilateral lesions diagnosed in the past three years than in the decade preceding. The statistics of Krönlein show 92 per cent of cases seen in the routine clinic to be unilateral. There are few statistics on this subject but the evidence goes to show that early tuberculosis is usually unilateral while late tuberculosis is more apt to be bilateral, except in children, where bilateral lesions are common. Kapsammer found in 20,770 autopsies that there were 191 cases of renal tuberculosis of which 67 were unilateral and 124 bilateral. Vigneron out of 250 autopsies found only 123 unilateral cases. Balfour, in the Mayo Clinic, found 80 per cent of the cases unilateral at operation; at autopsy 75 per cent bilateral. Morris found 9 unilateral cases in 72 autopsies.

In our series there were 40 right-sided lesions and 27 left-sided, while in 3 the side is not mentioned.

Family history of tuberculosis and definite history of exposure is positive in 10 cases, negative in 38, while in 22 this point was not recorded. There are no satisfactory data as to the conditions under which the patients live.

Duration of symptoms. The average duration of symptoms before operation is 2 years and 6 months, the shortest is 3 weeks and the longest 10 years. Of the 70 cases, only 24 claimed the benefits of operation before one year from the date of appearance of symptoms. This fact may and undoubtedly does have, through extensive bladder involvement, considerable significance in the many cases of troublesome bladder symptoms which have persisted over long periods of time.

In the earliest cases operated upon, even

one case in which there had been but 3 weeks of symptoms preceding operation, a well-marked lesion undoubtedly existed for a considerable time before the onset of symptoms and during this period was absolutely quiescent. It is not known what change takes place in the nature of the lesion at the onset of symptoms, but it is most probable that pyelitis with resulting bladder irritation and infection is the explanation.

Presenting symptom. The presenting symptom, i.e., the symptom which first brought to mind the fact that there was some abnormality of the body, shows that bladder irritation was by far the commonest of early symptoms, with pain in the kidney region on the affected side second. Below is given a table of the relative frequency of the presenting symptoms:

	No. of cases	Per cent
Bladder symptoms (bladder pain, frequency, burning, tenesmus, etc.)	34	48.5
Pain in the kidney region	25	35.7
Loss of weight and strength	3	....
Pain in penis on micturition	2	....
Hæmaturia without any other symptom for a time	3	....
Chills and fever with pyuria (complicated tuberculosis)	1	....
Referred pain (colic) associated with hæmaturia	1	....
Acute retention from pus and blood-clot	1	....

The facts recorded are not sufficiently detailed to allow of extensive generalization as to the character of pain in renal tuberculosis. The data available indicate that pain in the kidney region, whether occurring as presenting symptom or in the course of the disease, is of dull, aching character, situated in the costovertebral angle or indefinitely located in the affected side, except in the few cases in which there is hæmorrhage with the passage of blood-clot down the ureter, in which cases the character of the pain becomes colicky and is localized and referred as in ureteral calculus. There were no cases of crossed pain, i.e., referred to the unaffected kidney.

In this series of cases, dull, aching pain in the kidney region occurring at some time in the course of the disease is recorded in 38 instances or 54.2 per cent; sharp pain in kidney region in short attacks, 10 cases, 14.2 per cent; ureteral colic associated with hæmaturia, 11 cases, 15.7 per cent. Bladder pain bears a close relation to other bladder symptoms such as frequency and tenesmus and its frequency of occurrence is that of acute bladder symptoms.

General symptoms of loss of weight, anæmia, loss of strength, anorrhexia, and an occasional feeling of flushing (temperature) but usually not in marked degree, were observed in three-fourths of the cases.

CLINICAL SIGNS

Temperature. In two-thirds of the cases (46) the temperature fluctuated irregularly between normal and 100°. What cultural evidence is available indicates that these were not cases complicated by secondary infection. In 2 cases with secondary infection the temperature rose to 102° to 104° and remained so until operation, over a period of 5 and 7 days respectively. In 20 of the cases the temperature was normal. In the remainder of the cases, 3 in number, operation followed immediately on entrance into the hospital.

The urine. Sixty-one cases or 87.1 per cent of the cases showed from a trace to the very slightest possible trace of albumin. Only 2 showed none at all, but this was on a single examination. In the remaining 7 no urine analysis had been recorded.

The sediment showed pus in 60 (85.7 per cent) of the cases and microscopic blood in 33 (41.1 per cent). Twelve other cases gave a satisfactory history of having passed blood at some time since the onset of symptoms. In 2 cases a sediment was not done since there was a negative test for albumin, a procedure which is indefensible.

The amount of pus present was sufficient to lead the house officer to describe the urine as cloudy in 38 (55 per cent) of the cases. Blood was macroscopic in but 10 of the cases. The total number of cases in which there was macroscopic or microscopic blood or a satisfactory history of bloody urine at some time since the onset of symptoms was 45 cases or 64.2 per cent of the 70 cases studied.

Casts. Four cases showed casts. Three of these could not be traced. The fourth

reported 8 years after operation, having in the mean time developed an epididymitis (tuberculous). His urine still showed a large number of granular casts but he had no œdema and his blood pressure was 150 mm. He had no complaints other than a little frequency. Guinea pig inoculation was negative. Lesions of this nature must be considered toxic and have their origin in the by-products of growth of the infecting organism. The fact that the lesion has not progressed in this case in spite of increased work being thrown upon the remaining kidney leads one to conclude that the progress of the disease ceased after nephrectomy.

OTHER TUBERCULOUS LESIONS

Lung tuberculosis. Thirteen cases (18.5 per cent) in the series showed definite clinical and in some cases X-ray evidence of consolidation with or without evidence of activity of the process, or gave a history of unexplained pleurisy. Six of these cases are included in the list of dead under distant mortality. One patient has arrested tuberculosis of the lungs and is now under treatment at a sanitarium. Of the 5 known to have died, there were no necropsies but the city clerk's record shows entry of "death from tuberculosis."

The frequency of occurrence of pulmonary tuberculosis in patients harboring renal tuberculosis is difficult to estimate since few cases operated upon die in the hospital, where, by post-mortem findings, the presence of lung involvement can be demonstrated. The following interesting observations have been recorded.

Braasch finds that 6 per cent of the cases of renal tuberculosis at the Mayo Clinic had demonstrable lung involvement. Wildbolz found among 76 cases of renal tuberculosis apparently cured by operation only 8 which showed tuberculosis outside the genito-urinary tract; 5 of these were phthisis and 3 adenitis. Israel found that lung tuberculosis was the cause of 45.2 per cent of all late deaths. In Asakura's series all late deaths are ascribed to lung tuberculosis. Kelynack reported (1897) that in autopsies studied with reference to renal tuberculosis 80 per cent had lung tuberculosis. Von Frisch found old lung foci in all cases operated in his series. Walker from the Johns Hopkins Hospital reports renal tuberculosis in 23 cases out of 482 autopsies performed on patients dead from pulmonary tuberculosis. In these cases the diagnosis of renal involvement was not made since the patients gave no history of renal symptoms. Tilden Brown from the Presbyterian Hospital, New York, reports 68 cases of death from phthisis, of which 23 had renal involvement.

It is extremely doubtful if early lesions are recognized and it is almost certain that we miss tuberculosis of the bronchial glands in many cases; however, there is no reason to believe that early tuberculosis of the lungs contra-indicates operation.

Tuberculosis of the epididymis was found previous to operation in 3 cases. In the 2 instances in which it was unilateral it occurred on the same side as the kidney lesion. The other case had bilateral epididymitis but the record does not state which side was involved first.

Prostatic tuberculosis. There is insufficient data in our records as to involvement of prostate and seminal vesicles to allow of any conclusions as to frequency. In many instances there is no rectal examination recorded; particularly is this true of the earliest cases, at which time histories were less complete. One case showed tuberculosis of the prostate with negative vesicles. One case was diagnosed as genital tuberculosis without specifications as to what organs were involved. In 14 cases the rectal examination is recorded as negative.

Tuberculosis of the knee was found in 3 cases (4.2 per cent) of the series. In none of these was the process active. Braasch reports 7 per cent joint tuberculosis, 3 per cent active.

Tuberculous adenitis in the glands of the neck was found in 2 cases. They were not active at the time the renal symptoms developed.

Tuberculous stricture of the urethra was found in one case associated with prostatic involvement. The stricture was long, extending from the penoscrotal angle backward to the end of the bulb. This patient

died and was probably a bilateral case at the time of operation.

In all there were 24 "other lesions," i.e., lesions other than the kidney, ureter, and bladder. They occurred in 19 patients. The percentage of patients suffering from other demonstrable tuberculous lesions at the time of operation is 27 per cent.

OPERATION

In a few of the early cases there had been a previous nephrotomy done for tuberculous pyonephrosis or for "perinephritic abscess." All the cases were subsequently treated by nephrectomy and are properly included in this series. The date of the nephrotomy previous to the nephrectomy averaged for the 5 cases 3 years. In 4 there were sinuses, the other was a healed wound at the time of nephrectomy.

In all cases a nephrectomy was performed with a partial ureterectomy, with varying lengths of ureter removed. The wounds were all drained and no especial effort was made to sterilize the ureter.

KIDNEY LESION

The nature of the kidney lesion and the relation of the type of lesion to recovery of the patient is shown in the table:

	Cured	Unimproved	Dead	Total
Tuberculous pyonephrosis	14	4	7	25
Autonephrectomy calcified kidney	2	..	2	4
Early tuberculosis (usually single cavity)	4	3	3	10
Tuberculous nephritis	2	..	..	2
Cases diagnosed "Tuberculosis" (extensive involvement)	10	2	7	28
Ureter thick, evidently tuberculosis. Normal looking kidney	1	..	..	1
Total	42	9	19	70

From the above figures it is apparent that in extensive tuberculous involvement, even in tuberculous pyonephrosis, recovery is the rule, while in early tuberculosis the percentage of cured cases is low.

The relation of pain as a symptom to the character of the pathological lesion is difficult to interpret, yet some interesting facts are presented. In all the cases (25) in which the lesion found was pyonephrosis, there was a history of renal pain. The remaining 13 instances where this symptom was mentioned were about equally divided between the "early lesions" and "extensive lesions."

The occurrence of pain in pyonephrosis can be properly considered as due to distention. In the "early lesions" and in the 25 instances in which pain was the presenting symptom of the disease, explanation of the symptom on mechanical grounds becomes difficult unless it be due to renal congestion taking place during early stages of infection.

The average duration of time since operation for the series of 70 cases, excluding the 4 immediate deaths, is 5.6 years; for the cases considered cured, 6.2 years; for the unimproved, 4.4 years. Other facts concerning the time element in convalescence will be discussed at another time.

Some interesting data concerning the relation between duration of the disease before operative intervention and the course of convalescence develop from a detailed study of the series. Early lesions in young individuals as a rule have done less well. This observation was borne out particularly in some of our recent cases, not included in this study. Two factors seem to be significant in this connection; one, acquired resistance of the patient and the other, the extent of active perinephritic involvment. Some patients seem to acquire almost no resistance and the disease progresses in an uninterrupted downward course. These patients are not benefited by operation. While some cases which have been operated upon early in the disease have made rapid recovery—one in particular on whom nephrectomy was done in the third week of symptoms was free from symptoms immediately after operation and has remained well since—recovery is not the rule. In other cases in which the symptoms have run 3 to 4 years before operative intervention there has resulted a marked downward course to the disease similar to that of the early cases with marked perinephritis. These cases seem to have little or no resistance to the disease, and nephrectomy does not produce the desired effect of enabling the patient to care for whatever disease is left behind at nephrectomy.

The average age of the cases recorded as

dead or unimproved is 28 years with but 6 cases over 30 years of age and all under 40. All the patients who had passed the thirtieth year had more or less extensive "other lesions" and would properly be considered as having low resistance. The duration of symptoms before operation for the "dead" is 1.5 years; for the "unimproved," 1.8 years; while that for the "cured," it is 3.2 years.

In the face of the above facts, as far as the evidence of this series can influence our opinion, it would seem advisable to select cases for operation and perhaps to wait in some for immunization to take place before performing a nephrectomy. Much more information is essential before such a generalization can be made and there remains but one course in treatment, i.e., nephrectomy, as soon as the diagnosis can be made. The factor of resistance cannot be measured and there are young patients who do well under early nephrectomy.

URETER

The condition of the ureter is not given in a large number of the cases of this series. The kidney condition apparently occupied the entire field of vision of the surgeon. The condition of the ureter was not mentioned in 52 of the cases. In 18 it is recorded as thickened while there were no cases in which a normal ureter was reported. In our later cases in which especial emphasis was placed on the condition of the ureter we have been unable to find a single case in which definite tubercle formation was not demonstrated where enough of the ureter was examined. In one case with a single lesion of the upper pole of the kidney and in which the duration of bladder symptoms was short there was but one tubercle found in an otherwise apparently normal ureter.

There is no evidence of any examination of the ureter for stricture.

ANOMALIES OF THE KIDNEY

Harbitz and others have pointed out the occurrence of tuberculosis in abnormal developments of the kidney. We have found no anomalies of the kidney in this series.

END-RESULTS

Immediate mortality. Of the 103 cases operated on during the period covered in this review, there were four deaths taking place in the hospital as a result of operation. This gives an immediate mortality of 3.8 per cent. In comparing this result with that of other observers, it must be remembered that in some reports, immediate mortality is made to include those deaths taking place within the first 3 or even 6 months after operation. Immediate mortality is properly considered to be death as the result of operation and taking place in the hospital where the patient is under observation. This, I believe, is now the accepted interpretation.

The cause of death in the 4 cases was as follows: One patient died the seventh day after operation of pneumonia (necropsy obtained). One patient died the eleventh day of pyelonephritis of the lone kidney (necropsy). Two patients died immediately of hæmorrhage and shock, both being in poor condition at time of operation, one having had secondary infection of the tuberculous kidney.

Immediate mortality has improved with better diagnosis and operative technique.

Brodeur up to 1890 had 58 per cent mortality in nephrectomy for tuberculosis; Palet up to 1893, had 40 per cent; Pousson up to 1902, had 28.4 per cent.

In comparison with this the mortality of Albarran, Barth, Caspar, Cathelin, Czerny, Dollinger, von Frisch, von Illyes, Israel, König, Krönlein, Küster, Nicolich, Pousson, Rafin, Recsey, Rovsing, Rotter, Schede, Tuffier, Wildbolz, and Zukerkandl, in the years 1902 to 1908 for 847 cases operated was 11.1 per cent. Wildbolz reports his immediate mortality for the years 1902 to 1914 for 139 cases at 2.8 per cent. Asakura for the years 1905 to 1910 had 5.7 per cent of deaths. Israel's collection of 1,023 cases reported in 1911 had a mortality of 12.9 per cent. Von Frisch reported in 1911 a series of 100 nephrectomies with a 10 per cent mortality. Braasch in 1912 reported 203 nephrectomies with a mortality in the hospital of 2.9 per cent.

It seems safe to expect that in the hands of good surgeons at the present time immediate mortality will be below 5 per cent.

CAUSES OF DEATH IN EARLY MORTALITY

The causes of death in the course of convalescence after nephrectomy for tuberculosis in a collection of cases reported from various clinics show that the commonest factor is pneumonia, the second in frequency "shock." Ether is the anæsthetic in the cases of death reported from pneumonia in which the anæsthetic is mentioned. The common causes of death immediately after nephrectomy are as follows:

Pneumonia.
Shock.
Hæmorrhage at the time of operation.
Secondary hæmorrhage.
Reflex anuria with uræmia.
Acute nephritis in the remaining kidney.
Cardiac failure.
Necrosis of the small intestine in right-sided operation.
Trauma to the colon.
Endocarditis.
Miliary tuberculosis.
Thrombosis of the renal vein of the opposite side.
Pyelonephritis of the remaining kidney.

The absence of embolism as a cause of death after nephrectomy for tuberculosis is noteworthy. One would expect it to be of frequent occurrence in an operative procedure in which extensive manipulation of the kidney is necessary before the vessels can be ligated. I have not found recorded in the literature any case of death due to this cause.

LATE MORTALITY

Late mortality is defined as death from tuberculosis subsequent to temporary recovery from the operation. Fourteen of the cases of this series (20 per cent) are known to have died from tuberculosis. One patient on whom a nephrectomy was done for tuberculosis of the kidney, while not yet free from bladder symptoms 5 years after operation, was discovered to have carcinoma of the uterus from which she died 2 years later. This death is not reckoned in the late mortality. Of the late deaths from tuberculosis the assigned causes are as follows:

Four died within three months from tuberculous cachexia.

Three died from genito-urinary tuberculosis and exhaustion; one in the third year and two in the second.

Two died, one in the first year and one in the fourth, of tuberculous meningitis.

Five died at an average of 3.2 years after nephrectomy. In all these cases the death certificate is signed "tuberculosis." Half of the 14 cases died during the first year, the other half at an average of 3.3 years; only one lived more than 5 years.

From a compilation of data from 2,000 cases in which the cause of death in late deaths is given, the commonest causes in the order of their occurrence are general tuberculosis, lung tuberculosis, tuberculous meningitis, and miliary tuberculosis. Both meningitis and miliary tuberculosis are more frequently met with in genito-urinary tuberculosis than in other forms of disease.

WOUND HEALING

Of the 70 cases no data could be obtained in regard to the healing of the wound in 17. Of the remaining 53 cases seen at some time during their convalescence, 75 per cent had sinuses of more than 2 months' duration or had abscesses develop at some later time after the wound had closed, while 25 per cent healed by first intention and remained solid. Secondary sutures were done for persistent sinus in 3 instances and these wounds healed quickly and remained solid.

It is interesting to compare the end-result of the drained wounds of this series with that of our recent cases in which the wound was closed without drainage after injecting salt solution. In these later cases there were 25 per cent of the wounds which remained tight while 75 per cent developed abscesses. Abscess development was usually 3 to 5 weeks after leaving the hospital. This fact would have encouraged us to close all wounds without drainage had it not been our misfortune to follow the cases in the out-patient department and see the final results.

EVIDENCE OF EXTENSION OF THE DISEASE AFTER NEPHRECTOMY

Thirteen new lesions occurring in 9 cases are recorded. They are as follows:

Tuberculosis of the epididymis	5
Active pulmonary tuberculosis	3
Tuberculosis of the prostate and vesicles	2
Tuberculosis of the larynx	1
Tuberculosis of the remaining kidney	1
Tuberculous stricture of the urethra	1

These cases, save one in whom all tuberculous processes are now apparently arrested after epididymectomy, are included among the dead or unimproved.

Genital lesions in males. The occurrence of lesions of the epididymis either before or after nephrectomy was noted in 11 cases.

Epididymitis on the same side as the kidney	4
Epididymitis on the opposite side from the kidney	2
Bilateral	3
Side not mentioned	1

Eleven of the 34 males, or 32.3 per cent, developed epididymitis either before or after operation.

There were no instances in this series of tuberculosis of the fallopian tubes in the female.

SYMPTOMS OF PATIENTS NOW LIVING

Frequency is still a complaint in 24 cases. In 4 of these patients the urine contained no albumin, negative sediment, and no growth in culture. The condition was demonstrated to be due to contracted bladder, the size of the bladder varying from 2 to 5 ounces. In the great majority of the cases frequency had greatly improved by the end of 2 years. Some improvement in the contracted bladders followed mechanical dilatation, but in none was the condition cured.

The great majority of cases in which there was little or no improvement at the end of 2 years continued to have troublesome symptoms indefinitely. Few patients have made noteworthy improvement after 4 years.

ALBUMIN

Sixteen cases showed no albumin or abnormal sediment. Twenty-five cases showed albumin in small traces. In 4 the albumin was in large amount. One of the four had much pus with casts and the other three had casts alone. One of the latter had casts previous to operation. In 6 cases no urine specimens were obtained. Seven cases complained of no urinary trouble yet the sediment showed microscopic pus and there was a trace of albumin. Closer inquiry revealed that the patients had nocturia and some frequency to which they had become accustomed.

The presence of small amounts of albumin and pus in the urine is not evidence of persistent active secondary lesions. The condition is obscure and cystoscopic and cultural evidence as well as guinea-pig inoculation show no cause for the urinary condition. These cases are properly considered cured of tuberculosis but are not free from the effects of the disease which has resulted in permanent damage to the genito-urinary tract.

In 3 cases, with no bladder symptoms, there was a growth of bacillus coli and the sediment showed pus.

WEIGHT

All cases report either a temporary or permanent gain in weight and improvement in general health. Forty-four now weigh an average of 23 pounds above the pre-operative weight. The great majority have maintained the greater part of their gain. The largest gain is that of a woman who weighed 78 pounds at the time of operation and who now weighs 156½ pounds. Gain in weight followed by loss of weight with the present weight below that at the time of operation is noted in but 3 cases. These cases are numbered among the unimproved and have positive guinea pigs.

One case, a woman of 45 years, was seen 18 years after operation. She had no symptoms after two years save slight frequency. Her urine is negative as is also the guinea pig inoculated from bladder urine. She has gained 55 pounds, has been married 10 years, has had 3 normal pregnancies, and pneumonia, typhoid, and scarlet fever. She is strong and well save for a little frequency due to a contracted bladder. Her bladder holds 5 ounces and resists attempts at dilatation. She finds it necessary to void every 2½ hours as she has acquired the water drinking habit and has a small bladder.

HERNIA IN SCAR

Was present in one case but was small and not sufficiently troublesome to demand interference.

GUINEA-PIG INOCULATION

Was done in all cases where the patient sent a specimen of urine or was seen in person. In 6 instances the guinea pig was positive.

CURED

We have classified as "cured" those patients in good general physical condition; with maintained gain in weight; without evidence of extension of the disease, or if new foci have developed they are now quiescent; with improvement of bladder symptoms and no evidence of active lesions in the genito-urinary tract. Sixty per cent of the cases in the series conform to these requirements.

UNIMPROVED

The term "unimproved" is applied to those patients who may or may not have maintained their post-operative gain in weight; whose general physical condition is below normal; who still have active secondary lesions of the genito-urinary tract or in whom the disease has progressed and the new lesions are active; and in whom nephrectomy has not afforded the expected retardation of the disease; 12.8 per cent of the cases are unimproved.

MENINGITIS IN GENITO-URINARY TUBERCULOSIS

Israel, Simmonds, and Müller have produced evidence to show that the incidence of meningitis in tuberculosis of the genito-urinary tract is greater than in other forms of the disease. Israel states that 12 per cent of the cases operated upon for renal tuberculosis die of meningitis. Simmonds examined 60 cases of genital tuberculosis as to the cause of death and found that 19 (30 per cent) had died from tuberculous meningitis while there were only 5 per cent of deaths from this cause in lung tuberculosis. Müller examined in the Vienna General Hospital for a period of ten years, cases in which the cause of death was tuberculosis to ascertain the frequency of meningitis. He studied 5,372 cases and found that 223 died of meningitis, which gives 4 per cent of all deaths in tuberculosis due to meningitis alone. There were 723 cases of tuberculosis of the urinary tract alone and 77 of the genital tract alone, with 13 deaths among these from meningitis (1.6 per cent). There were 105 cases of tuberculosis of the genito-urinary tract, with 23 deaths or 22.5 per cent of all deaths in the combined form of the disease. Deaths from this cause are usually remote from the operation.

CONCLUSIONS

1. Young individuals with early lesions of the kidney which usually show considerable cortical tuberculosis and perinephritis do less well after nephrectomy than those of more advanced age in whom, however, the disease is often more extensive. The reason appears to be the extent of acquired immunity.

The practicability of artificial immunization of these individuals demands consideration.

2. Male patients offer more difficult problems in treatment than female and the prognosis is less favorable. Genital lesions are uncommon in the female while 32 per cent of the male cases of this series show genital involvement.

3. Immediate mortality for this series is 3.8 per cent. The operation of nephrectomy for tuberculosis is not serious as regards death of the patient. The immediate mortality is that consequent to pneumonia, shock, and accident, and should not exceed 5 per cent in unilateral lesions.

4. Late mortality for this series is 20 per cent. A late mortality of 15 to 20 per cent should be expected; about 50 per cent of the deaths taking place within the first 2 years, and the remaining 50 per cent before 5 years where the lesion is unilateral.

5. Sixty per cent of our cases can be considered cured. Somewhere between 55 to 70 per cent of the cases operated upon for unilateral lesions can be considered cured of active tuberculous lesions at the end of 5 years. There remain, however, in about 35 per cent of the cases legacies of the disease in abnormal urine and persistent symptoms. Nephritis probably of toxic origin and secondary infections of the remaining kidney; irritable bladders sometimes due to cicatrices; traces of albumin and slight pyuria are among the commonest permanent effects of the disease.

6. In 10 to 15 per cent of the cases the progress of the disease will not be checked by nephrectomy. There were 9 cases (12.8 per cent) unimproved by operation in this series.

7. The results for this series of drainage of the wound after nephrectomy compare favorably with undrained wounds in our recent cases in regard to sinus formation. There is approximately 25 per cent which heal by first intention and 75 per cent in which sinuses develop irrespective of the method of wound closure employed. In undrained wounds sinus development is late and where the follow-up system is not carried out may give a false idea of the value of the treatment.

NEPHRECTOMY DURING PREGNANCY[1]

By LOUIS E. SCHMIDT, M.D., F.A.C.S., Chicago

OPERATIONS during the course of pregnancy are common. Operative interference even in the uterus and in the adnexa as well is not unusual. I wish to discuss particularly the removal of a kidney during pregnancy. There are many interesting features bearing on this subject which are worthy of consideration. The question was brought forcibly to my attention in March, 1913, when a patient presented herself with the following history:

Patient No. 60,599, Michael Reese Hospital, referred by Dr. H. Banga. M. W., age 28. Knows of no serious illness. Has had four children, the youngest being 14 months. All deliveries were normal and easy. She has not menstruated since last birth, before which the periods were irregular, 6 and 7 weeks, lasting 4 and 6 days, always accompanied by slight pain.

Present complaint. Possibly last labor was followed by a slight temperature. Eleven months ago frequent and painful urination commenced. Now she passes urine five and six times each night and every half hour during the day. The urine is often bloody, with clots, and frequently milky and thick. She has not been able to sleep, has complained of considerable pain in the lower abdomen, and of late the pain at end of urination has become unbearable. She believes she has had slight chills followed by fever. The last few months she has become weaker and unable to attend to the slighest work. She has been practically unable to eat and badly constipated.

Examination. Temperature previous to operation ranging from 99.5° to 101° F. White blood count from 8,000 to 15,000. Blood pressure 105. Hæmoglobin 60 per cent. Urine, large quantity of pus, blood-corpuscles; microscopically, red blood-corpuscles were always present. There were no casts but a large number of epithelial cells. Bacteriologically, colon bacillus was demonstrated, and bacillus tuberculosis was found repeatedly in stained specimens. Physically the patient was greatly emaciated and weighed 108 pounds. Her skin was dry and swarthy. Her heart and lungs were negative. The abdomen indicated a pregnancy of about the end of the fifth month. Palpation of left kidney showed that it was much enlarged and sensitive. The right side was negative.

Cystoscopic examination. Bladder mucosa congested. Left ureteral catheterization revealed obstruction at 2.5 cm. from orifice. The obstruction could not be passed and no urine was collected. Right ureter catheterized and catheter easily passed to pelvis. The urine on this side showed no abnormal elements. A phenolsulphonephthalein test gave on left side appearance in 4.5 minutes; 17 per cent first 15 minutes and 19 per cent second 15 minutes. No dye could be detected on left side. X-ray examination showed much enlarged kidney as well as ureter. The right side was negative.

Operation, May 22, 1913. Incision from tip of last rib on left side extending outward and downward toward the spine of the pubes. The kidney and ureter were exposed. Both were markedly adherent to all surrounding parts. The ureter was tied off below the obstruction, the vessels to the kidney were separately ligated, and the fatty capsule with kidney and ureter were removed *en masse.* The wound was closed, except for drainage at both ends of incision. After operation the temperature rose to 105°F., following slight chill. Otherwise there were no untoward symptoms. The patient made a slow recovery. At the lower angle drainage persisted until following childbirth.

On September 11, 1913, Dr. Simon attended normal delivery. The baby died twelve days later of sepsis. The mother was discharged from the hospital in January 1914.

Pathological report made by Dr. Kirk of Michael Reese Hospital. On gross examination, the kidney

[1] Read before the American Association of Genito-Urinary Surgeons, White Sulphur Springs, West Virginia, May 18–20, 1915.

is slightly larger than normal, measuring 12 cm. from pole to pole. It weighs 270 grams. The surface is irregular and nodular, and the capsule is thickened. The ureter is attached. It is considerably thickened, congested, and inflamed. The distal 3 cm., of the ureter is hard, firm, resistant. On cut section, made by incising the kidney longitudinally, there are four large pus cavities measuring 2 to 4 cm. in diameter; two at each pole. These are filled with a caseous material of the consistency of putty, upon the removal of which, ragged-walled cavities are left. About the cavities, there is an increase in the connective tissue.

Diagnosis. Tuberculosis of the kidney, pyonephrosis, ureteritis.

In looking through the literature, I find that Hartmann in *Traveaux de Chirurgie*, 1913, refers to 30 cases, in which nephrectomy was performed during pregnancy. I have found five more cases and have included them all in the series of cases reported at the end of this article. The following facts were deduced from a study of the cases:

1. Total number of cases, 35.
2. Mortality, mother 2 cases, 5.7 per cent.
3. In seven cases the effect on pregnancy is not indicated. In one case the operation was done almost at the time of delivery and spontaneous birth with living child occurred. Of the remaining cases 21, 77 per cent, had normal labors at term. Three had spontaneous abortion, one had induced abortion, one had induced labor, one had dead fœtus extracted.
4. In only four cases, 15 per cent, it is stated positively that harmful effects on the fœtus occurred.
5. As regards the time of the operation.

In the second month of pregnancy	4 cases
In the third month of pregnancy	6 cases
In the fourth month of pregnancy	8 cases
In the fifth month of pregnancy	7 cases
In the sixth month of pregnancy	4 cases
In the eighth month of pregnancy	2 cases
Not definitely stated	4 cases

Even since the first nephrectomies have been carried out, it has been satisfactorily demonstrated, and more convincingly every year, that the remaining kidney compensates for the loss of its mate, becomes hypertrophied, and answers every purpose of the individual.

There is in connection with this subject another angle of equal, if not greater, importance; namely, that a great number of women on whom a kidney has been removed for one cause or another later become pregnant. From the literature as well as from personal observation, I am inclined to believe that, if pregnancy occurs not too soon after nephrectomy, it is not hazardous provided the remaining kidney is not diseased. However, if the remaining kidney is diseased and not functionating properly it is dangerous to mother and child. To illustrate: if nephrectomy has been performed in a patient suffering from congenital cystic kidney, in which cases it is a fairly well established fact that the opposite kidney is always of the same type, pregnancy certainly is detrimental to the mother and forbodes abortion or death to the fœtus. In case of pregnancy following nephrectomy for tuberculosis, provided there is no tuberculosis of the bladder or ureter, or for stone and infection, no harm can result. Marriage should not be sanctioned where disease of the bladder, such as tuberculosis, is present, or if the remaining kidney is diseased.

I can recall several instances where pregnancy went to term following nephrectomy, without detriment to mother and child. When we consider that the kidneys are frequently involved during pregnancy and that degenerative changes and various types of nephritis, as well as pyelitis and pyelonephritis, make their appearance at such times, it will be understood that during pregnancy the kidneys are unquestionably put to a severe test, and for some reason easily become involved. Therefore after pregnancy a woman in health must be kept under careful observation, and to some extent, as Tietze and others have pointed out, a woman with only one kidney runs a greater risk. But to remove a kidney during the course of pregnancy and throw all the work on to one is undoubtedly a greater strain on the functional capacity than in the mentioned instances. Therefore, I believe that it must be carefully considered whether or not a nephrectomy during the course of pregnancy is to be advised. It has been shown that toward the end of pregnancy, the combined nitrogen is increased, unquestionably indicating that greater demands are made on the

healthy kidneys. Particularly toward the last month of pregnancy albumin, with or without the presence of casts, is frequently noticed. There is a slight difference in percentage as regards primipara and multipara. These cases can and often are freed from albumin and casts by either diet or normal termination of pregnancy.

There is, however, a far more grave type of albuminuria which is accompanied by nausea, vomiting, œdema, and other signs and symptoms, and which necessitates the interruption of pregnancy in order to save the life of the mother. Oftentimes spontaneous abortion occurs. In contradistinction to the other form, this type continues even if the mother recovers. In both instances it is pregnancy, the product of pregnancy, or a toxæmia which is the cause.

Therefore, to do a nephrectomy during the course of a pregnancy involves the question whether in cases in which a diseased kidney has already influenced its mate the great problem of eclampsia will not appear. I believe that it is now the general opinion, that in the vast majority of instances of eclampsia a chronic disease of the kidney exists before pregnancy. In recent years the literature has teemed with the problem of the infections—I have particular reference to pyelitis and pyelonephritis. I do not wish to discuss the mode of access. Suffice it to state that these conditions are not at all uncommon. It must be admitted that either one or all of the aforementioned conditions are frequent occurrences whether the woman be free from all kidney disease or even if a chronic disease of the kidney exists, before pregnancy. Naturally if a woman has only one kidney, it is a matter of greater import and it follows that if a nephrectomy is carried out during pregnancy, not only the above facts must be considered, but the sudden transference of all work to the remaining kidney adds greatly to the problem. I might state that if the remaining kidney is diseased the problem is still of greater import to both mother and child.

I can still recall the time when the consent of the obstetrician to marriage was grudgingly given in those cases where it was known that one kidney had been removed for one cause or another. I do not wish to pass over this subject too lightly, but I believe that I have pointed out some facts which must be taken into consideration, which, however, are appreciated more keenly than formerly. It is needless to state that there are many authorities that still hesitate in giving their consent to marriage under such circumstances. One must consider undoubtedly the cause for which the nephrectomy was carried out. With all our modern diagnostic methods at hand it is certain that one is in a much better position to judge correctly than a decade ago. This question, as well as the one under consideration is intertwined with the same problems and hence I have taken the opportunity to express my views on both. I have collected in all 36 cases including my own and have already mentioned some of the more important facts. I do not wish to go into the details as to the methods of diagnosis employed, nor the question of the examination of the functional capacity of the kidneys, nor of the course of the patient after termination of pregnancy, for the simple reason that the cases recorded are not uniformly reported. However, my impression is that whenever conditions are present before pregnancy which demand nephrectomy, it is more desirable to remove the kidney before impregnation than to be obliged to carry out nephrectomy during pregnancy.

From the literature as well as from personal observation, I have come to the conclusion that a woman with one healthy kidney, and that implies that no infection remains whether it be tuberculosis or a pyogenic infection, does not carry much greater risk, nor does the fœtus, than the more fortunate woman who has two kidneys, and that consent to marriage and pregnancy cannot be withheld. There are questions such as how soon after nephrectomy may one, without danger, become pregnant. I am of the belief that if a period of several years of complete absence of kidney disease elapses it is safe. There is a greater need for exact observation of these cases, so that in case of the recurrence of any (nephrictic) symptoms, induced abortion or labor can be carried out in order to prevent

chronic kidney changes which might result in making an invalid of a woman having only one kidney.

The following is a series of cases in which nephrectomy was performed during pregnancy.

CASE 1. Reported by Andrews in the *Journal of Obstetrics and Gynæcology of the British Empire*, 1909, xvi, 249. The patient, aged 28, a multipara, four months pregnant, was operated upon for movable kidney and hydronephrosis—nephrectomy; recovery. Four years later she was operated upon by the same surgeon for unilocular cyst. The patient went to term without complications and has had five subsequent pregnancies.

CASE 2. Reported by Bickel in the *Indian Medical Record*, February 21, 1900, p. 172. The patient, a multipara, aged 29, was in the third month of pregnancy, when she was operated upon for hydronephrosis. Her recovery was delayed by a febrile dermatitis, but she was in perfect condition five months after operation. She went to term without complications, and labor was normal.

CASE 3. Reported by Braatz, in the *Deutsche Zeitschrift für Chirurgie*, vol. xlviii. The patient, aged 33, was in the eighth month of pregnancy. She was suffering from an epithelial tumor of the kidney pelvis, with complications. A nephrectomy was done, and the patient left the hospital in good condition. Pregnancy apparently went to term but there are no particulars given by the author.

CASE 4. Reported by Brown in the *New York Medical Journal* of August 16, 1890. The patient, a multipara aged 26, in the second month of pregnancy, suffered from chronic pyelitis, with severe hæmaturia simulating calculus, for which nephrotomy and nephrectomy were done. The patient recovered but aborted on the ninth day after operation.

CASE 5. Reported by Cooa in the *Annali di ostetricia*, 1903, xxv, 694. The patient, a multipara aged 35, in the third month of pregnancy, suffered from calculus and cystic kidney. Nephrectomy was performed and she left the hospital after six weeks, in good condition. Pregnancy proceeded to term without complications, and a healthy child was born. The patient subsequently became pregnant, with normal labor and lactation.

CASE 6. Reported by Cragin in the *American Journal of Obstetrics*, 1898, xxxiii, 36. The patient, a multipara, aged 25, was eight and one half months pregnant. She had an ectopic kidney blocking the pelvis, and hydronephrosis. Nephrectomy was done per vaginam. The patient recovered. The day following the operation labor started spontaneously and a healthy, living child was born.

CASE 7. Reported by Cumston, in the *New York Medical Journal* in 1902. The patient a multipara, aged 29, was in the fourth month of pregnancy. She suffered from pyelonephritis for which nephrectomy following nephrotomy was done. The patient recovered three weeks after the operation. She went to term without interruptions, and the child was born in good condition, without complications, at term.

CASE 8. Reported by Dobson, in the *British Medical Journal*, April 15, 1911. The patient, in the fifth month of pregnancy suffered from multiple abscesses of the kidney. Nephrectomy was done and the patient recovered. The effects of the operation on the pregnancy are not stated, but apparently labor proceeded to term, uninterrupted.

CASE 9. Reported by Gerster in the *New Yorker medizinische Monatsschrift*, 1897. The patient, a multipara, aged 26, was in the sixth month of pregnancy. She suffered from an echinococcus cyst of the kidney, for which nephrotomy followed by nephrectomy was done. She recovered, but aborted a month after the operation.

CASE 10. Reported by Harrigan in the *American Journal of Obstetrics*, 1914, lxix, 500. The patient, aged 21, was in the fourth month of pregnancy. She suffered from an acute infection of the kidney, for which nephrectomy was done. She recovered. The patient was delivered by induced labor and both mother and child are well.

CASE 11. Reported by Hartmann in *Traveaux de chirurgie*, fourth series, 1913, p. 455. The patient, a multipara, aged 34, was in the second month of pregnancy, when nephrectomy was performed for pyonephrosis. She recovered and went to term without any complications. She has had two subsequent normal pregnancies.

CASE 12. Reported by Hartmann (Pillet) in *Traveaux de chirurgie*, fourth series, 1913, p. 455. The patient, a multipara, aged 23, in the sixth month of pregnancy, was suffering from renal tuberculosis, for which nephro-ureterectomy was done. The patient left the hospital in good condition twenty days after the operation. She was delivered of a living, healthy child, at term. She had some slight vesical symptoms still, six months later.

CASE 13. Reported by Helferich in *Thesis of Blanc* (Paris), 1908, p. 74. Nephrectomy was done for pyonephrosis and the patient recovered. She went to term and was delivered of a healthy child.

CASE 14. Reported by Israel in the *Berliner klinische Wochenschrift*, 1897, xxxiv, 104. The patient, aged 20, primipara, was in the fifth month of pregnancy. Nephrectomy was performed for pyonephrosis and the patient recovered. Pregnancy proceeded to term without interruptions.

CASE 15. Reported by Israel in the *Archiv für klinische Chirurgie*, 1894, xlvii, 392. The patient, aged 20, in the fourth month of pregnancy, was operated upon for renal tuberculosis—nephrectomy, with recovery. Labor proceeded to term without disturbances and a healthy child was born.

CASE 16. Reported by Jarman, in the *Annales de gynécologie et pédiatrigue*, 1900, xiii, 662. The patient, aged 31, a multipara, was in the fifth month of pregnancy, when she was operated upon (nephrectomy) for tumor of the kidney. She recovered, and pregnancy proceeded to term without complications, and she was delivered of a living child.

CASE 17. Reported by Kosinski in the *Medycyna*, 1893, No. 40, and *Centralblatt für Chirurgie*, 1893, xx, No. 51. The patient was four months pregnant when nephrectomy was done. She recovered and pregnancy proceeded to term without interruption.

CASE 18. Reported by Krönlein in the *Verhandlungen der deutschen Gesellschaft für Chirurgie*, xxviii. The patient, aged 38, was in the third month of pregnancy, when nephrectomy for multilocular cystic kidney was done. The recovery was perfect, but the effects on the pregnancy are not given.

CASE 19. Reported by Landau in the *Deutsche medizinische Wochenschrift*, 1893, xix, 560. The patient was in the second month of pregnancy when nephrectomy was performed for kidney calculus. She recovered but the effects of the operation on the pregnancy are not given.

CASE 20. Reported by Lenger, in *Presse Médicale*, 1896, ci, 607. The patient, aged 29, primipara, in the sixth month of pregnancy, was operated upon (nephrectomy) for hydronephrosis. Eclampsia started the day after the operation, and the patient died on the third day after operation. A dead fœtus was extracted.

CASE 21. Reported by Zohmer, *Dissertation*, Griefswald. The patient, aged 37, multipara, in the fifth month of pregnancy, was operated upon (nephrotomy and nephrectomy) for pyonephrosis. She recovered, the pregnancy was not interrupted, and a healthy, well-developed child was born.

CASE 22. Reported by Legneu, *Bulletin et mémoires de la Société obstétricale et gynécologique de Paris*, May 1904. The patient, aged 27, multipara, two and one half months pregnant, was nephrectomized for kidney abscess. She recovered, but suffered from tuberculous pleurisy some months later from which she recovered. The pregnancy continued normally and a healthy child was born in the hospital, at term.

CASE 23. Reported by Marz, *Prezglad Leharski*, 1901. (Quoted by Coon, *Annales di ostetricia*, 1903, xxv, 698.) The patient, in the fifth month of pregnancy was operated on for tumor of the kidney. She recovered. The effect on pregnancy is not given, but apparently it proceeded normally to term.

CASE 24. Reported by Mirabeau, *Archives für Gynakologie*, 1907, lxxxii, 484. The patient aged 34, multipara, in the fifth month of pregnancy, was operated upon for tuberculous kidney (nephrectomy). She recovered, went to term, and a vigorous child was born without complications. The patient continued in good health for several years after the operation.

CASE 25. Reported by Martin, *Zeitschrift für Geburtshilfe*, 1890, xviii, 375. The patient, aged 22, length of pregnancy not definitely stated, was operated upon (nephrectomy) for movable kidney and hydronephrisis. She recovered. Apparently the pregnancy was not affected but the reporter does not state the facts in this regard.

CASE 26. Case of Nicolich, reported by Hartmann in *Traveaux de Chirurgie*, fourth series, 1913, p. 458. Particulars as to the age, and length of pregnancy are not given. The patient was operated upon for tuberculosis of the kidney—nephrectomy. She recovered and labor was normal, at term.

CASE 27. Reported by Parkes, *American Journal of the Medical Sciences*, September, 1890, c, 264, Case 23. The patient, aged 26, multipara, in the fourth month of pregnancy, was nephrectomized for adenoma of the left kidney. Recovery was complete in two months. She went to term, and a well-developed child was born.

CASE 28. Reported by Pilz, *Zentralblat für Gynakologie*, 1905, p. 649. The patient, aged 27, in the fourth month of pregnancy, was nephrectomized for renal tuberculosis. She recovered and pregnancy was not interrupted.

CASE 29. Reported by Polk, *American Journal of Gynecology and Obstetrics*, 1898, xiii, 164. The patient, aged 35, multipara, in the fifth month of pregnancy was nephrectomized for hydronephrosis. She recovered and labor was normal, at term.

CASE 30. Reported by Prochownick (quoted by Hartmann in *Traveaux de Chirurgie*, fourth series, 1913, p. 458). The particulars as to age and length of pregnancy not given. Nephrectomy was performed for multilocular cyst of the kidney. She recovered and labor was normal, at term.

CASE 31. Reported by Rudolph, *Dissertation Griefswald*, 1898. The particulars not given as to age and length of pregnancy. Nephrectomy was performed for renal suppuration. She recovered but the effect on the pregnancy is not given.

CASE 32. Reported by Scudder, *American Journal of the Medical Sciences*, 1895, cx, 646. The patient, aged 22, primipara, in the third month of pregnancy, was nephrectomized for a carcinomatous tumor of the kidney. She recovered, went to term, and a living child was born without complications.

CASE 33. Reported by Tuffier, *Annales des maladies des organes génitourinaires*, 1892, x, 413. The patient, aged 30, multipara, in the third month of pregnancy, nephrectomized for severe traumatic kidney lesions and suppurative nephritis. After fourteen days the patient suffered from phlegmasia alba dolens, and died two days later (iliac thrombosis). Fourteen days after the operation the patient aborted.

CASE 34. Reported by Twynam, *British Medical Journal*, 1898, i, 423. The patient, aged 32, multipara, the third month of pregnancy, was nephrectomized for pyonephrosis. She suffered a light

attack of uræmia which subsided and the patient recovered. Abortion was induced one month after operation, as complications were anticipated.

CASE 35. Reported by Walmar, *Dissertation, Halle*, 1900, p. 16. The patient, aged 28, in the fourth month of pregnancy, was nephrectomized for hydronephrosis. She recovered, pregnancy was uninterrupted, and delivery normal.

CASE 36. Reported by Schmidt, before the American Association of Genito-Urinary Surgeons in 1915. The patient, aged 28, multipara, in the fifth month of pregnancy, was operated upon because of tuberculosis of the kidney and ureter and a ureteronephrectomy was performed. She recovered, pregnancy was uninterrupted, delivery normal, but the child died 12 days later of sepsis.

A CASE OF DIETL'S CRISIS IN AN ANOMALOUS RIGHT KIDNEY ASSOCIATED WITH A LEFT PELVIC KIDNEY[1]

BY R. C. BRYAN, M.D., F.A.C.S., RICHMOND, VIRGINIA

BILATERAL congenital dystopia is relatively infrequent. Strube in *Virchow's Archives* of 1894 mentions but four cases. The following are the available reported cases.

Armstrong[2] reports the following case:

Male, age 45, native of Germany, admitted to Memphis City Hospital, October 14, 1886, in comatose condition, who died in a few hours. The autopsy showed the upper border of right kidney upon twelfth rib, its lower border extending below the crest of the ilium. The gland was normal in size, slightly movable, and the blood-vessels except of lower origin, were normal. The upper border of the left kidney rested on the first sacral vertebra, and the gland rested on the sacrum. The left renal artery arose at the bifurcation of the aorta and entered the anterior external surface of the kidney about its center. The pelvis of the kidney was imperfectly developed on the posterior surface and contained the ureter and vein. The sigmoid flexure of the colon passed over to the right side, and the rectum was on that side between the internal border of the kidney and the pelvis. Both kidneys were pale; chronic nephritis.

Dr. Lenzen Belcke[3] reports in extensive detail a case of dislocation of both kidneys as the result of a fall from a wagon wheel to the ground. The patient struck his back upon the hub of the wheel and the wagon tongue, and fell from here to the ground, striking firmly on the buttocks. He noted a severe pain in the back at the time and became unconscious for 10 to 15 minutes. Subsequent developments showed traumatic right wandering kidney. The patient declined operative fixation. He left the hospital much improved June 30, 1894. In January, 1897, he developed chills and fever and pain in the abdomen and back, similar to the symptoms he showed at the time of dislocation of the right kidney, only this time the pain was on the left side. In the interim the right mobile kidney had also given him trouble. Both kidneys were displaced downward to a considerable extent. The condition of the patient grew worse so rapidly that malignant disease of the left kidney was suspected. Surgical measures had again been refused. Death October 29, 1897. The writer is convinced that malignant disease of the left kidney had developed, but autopsy was also refused.

E. Meuschen,[4] St. Petersburg, reports the following case:

During autopsy on a female, age 19, who had died from pernicious anæmia, Meuschen found a bilateral renal dystopia. The left kidney as well as the right then lay normally. The left kidney lay immediately in front and to the left of the promontory and in front of the edge of the sacrum, continuous with the innominate line of the os ilium.

The lower pole of the left kidney reached into the small pelvis for 3 to 4 cm. . . . The right kidney, somewhat longer and narrower, lay entirely within the large pelvis, its lower pole just touching the prolongation of the linea innominata at the sacrum. Both kidneys were firmly imbedded in the surrounding tissues and were in no way connected with one another. The two kidneys were not normal in shape. The left kidney did not possess the oblong, bean-shaped form but was more rounded; its upper portion was narrower and broadened out below. The narrow frontal diameter was wider than usual, while the long frontal diameter was shorter than normal. Measurement of the anterior surface of the kidney showed a length of 8 cm., width 5.75 cm., and 4 cm. thick. The left kidney had the appearance of a rounded disk. It looked more like a potato; it showed no lobulation. On the anterior surface, a trifle above the center, was found the hilus to which the vessels descended from above,

[2] Congenital deformity of kidney; anomalous location of both kidneys. St. Louis Courier Med., 1887, xvii, 505.

[3] Ein Fall von Dislocation beider Nieren nach Unfall: Nephroptosis traumatica. Monatschr. f. Unheilk., 1898, v, No. 7, 15; p. 209.

[4] Ein Fall von beiderseitiger Nierendystopie. St. Petersb. med. Wchnschr., 1909, xxxiv, No. 41, 537.

[1] Read before the American Association of Genito-Urinary Surgeons, White Sulphur Springs, West Virginia, May 18–20, 1915.

terminating in a groove in the upper half of the kidney, while the ureter descended behind the artery and vein.

The right kidney looked more like a normal kidney, but was smaller, its thickness was in excess to the width and length in proportionate measure — it was 9.25 cm. long, 4 to 4.5 cm. wide, the lower pole being wider than the upper, and the thickness 3.5 cm. The right kidney did not lie so close to the spinal column as the left. The left ureter had thinner walls and a smaller lumen than the right.

The cadaver showed throughout decreased measurements in all the blood-vessels. Accessory kidney blood-vessels noted.

Bilateral dystopia of the kidneys, distinct hypoplasia of the kidneys, in size as well as development, increase and variation in blood-vessel supply. The kidneys were separate; in no way connected. The entire picture might be construed as a development inhibition, a restriction or absence of growth of the kidney along the wolffian body as described by Strube.

All the blood-vessels of the body showed a lumen smaller than normal, and all the arteries were underdeveloped. The left common iliac artery passed behind the kidney shortly after its origin, entered the adiposa capsule posteriorly, and divided in the lower third of the hypogastric artery. The right common iliac artery proceeded normally downward, passing the lower pole of the kidney obliquely. The aorta gave off on its anterior surface, about 1 cm. from the bifurcation, an artery for the left kidney. This left renal artery was a little smaller than a goose-feather stem, and divided behind its origin into two branches. At the point where the upper branch came near the pole of the kidney, it gave off a branch of about the same size which entered the parenchyma of the kidney at the upper pole. Both main branches disappeared into the hilus to the kidney. The lower branch was more median to the hilus and behind the ureter. These two branches had a left renal vein between them. Shortly in front of the point where the left renal artery divided into the two branches, the vein emptied into the left iliac vein.

The right kidney received from the right lateral surface of the aorta, about 2 to 3 cm. above the bifurcation, an upper right renal artery, which, horizontally coursed to the hilus of the right kidney, where it divided into two branches, the upper branch supplying the upper part of the kidney, the lower the more central portion. From the right common iliac artery, immediately below its origin, arose the right inferior renal artery, which also coursed toward the kidney horizontally approaching the hilus, behind the ureter. It also divided into two branches in the hilus behind the ureter; one for the upper pole and one for the more central portion of the kidney. In the hilus two veins united which emptied into the inferior vena cava.

Both suprarenal bodies lay in normal position.

A research of the literature at his command revealed only four cases of bilateral dystopia reported by Strube[1] in which the lower poles were not united. These cases were reported by Martin St. Ange,[2] Boyd,[3] Poirier,[4] and Potherat and Morderet.[5]

Karl Joseph[6] studied the records of the past 20 years of the pathological Institut at Giessen and found among other dislocations or displacements of the kidney, one case of bilateral dystopia of the kidney.

Male, age 19, fell from wagon and sustained fracture of the base. At autopsy no kidneys were found at the normal site, nor did any arteries emanate from the aorta at this point. The suprarenals were present on both sides of the spinal column at normal level. They were about the size of a "mark." The cæcum was not fixed, but was easily mobile; the vermiform appendix was unusually long, but showed no other pathological changes. Fixation of the colon began at upper pole of right kidney. The right kidney, 11 cm. long, 4 cm. wide, and almost as thick, rounded in shape, extended with its lower pole to the linea innominata. An artery emanated from 1.5 cm. above the site of bifurcation of the aorta, opposite the origin of the mesenteric artery inferior, and after a course of 4 cm. divided into three branches, all of which enter within the hilus of the kidney.

The left kidney, 9 cm. long, 6 cm. wide, 2 cm. thick, lay partly in the small pelvic, partly on the left psoas muscle and linea innominata; the hilus was directed forward and to the right. This kidney received its blood supply from two arteries; one arising immediately above the bifurcation of the aorta and immediately dividing into two branches. One of these again subdivides and both branches enter the kidney at the lower pole; the other branch runs along a twisted open groove of the kidney and then winds around to the front where it enters the hilus of the kidney. The second artery arises from the origin of the left common iliac artery; this is the larger and main artery of the kidney; its course is at first downward, and at the lateral margin of the kidney divides into two branches, one of which enters at the lateral margin of the kidney, while the other enters the hilus of the kidney by a circuitous route. The course of the veins was not described.

In discussion Joseph states that Strube reports 4 cases and Graser mentions 12 such anomalies.

[1] Virchow's Arch. f. path. Anat., etc., Berl., 1894, v., 137.

[2] Ann. d. sc. nat., ser. 1, v, 19.

[3] Med. Chir. Tr., Lond., 1841, 2. s., v. 6.

[4] Bull. Soc. anat., de Par., 1889.

[5] Bull. Soc. anat. de Par., 1889.

[6] Zur Kenntnis der congenitalen Lageanomalien der Niere. Dissertation, Giessen, 1910.

Tenchini[1] cites the following case:

Cadaver of female, age 60, died from double pneumonia in 1886, showed both kidneys in the pelvis on both sides of the sacrum. They were in contact with the rectum behind and the uterus in front. The patient had never given any sign of kidney disturbance during life, and the organs were normal and in normal position; nor were there any anomalies of the skeleton. The suprarenal capsules appeared to be normal. The left kidney had a very large hilus, situated anteriorly. The right, on the other hand, followed the median line and the ureters (shorter than usual but otherwise normal) emptied into the bladder separately. The left kidney had a large single artery arising from the primary iliac artery about 1.5 cm. from the terminal division of the aorta. After traveling for 7.5 cm. it joined the kidney, following a serpentine course for about 3 cm. and finally ending in the hilus. The right, on the other hand, had two renal arteries, both much smaller than the left and both arising from the common iliac, one (the upper) near the origin, the other near its termination. The first descended for a distance of about 6 cm., then it divided into one branch which entered the upper part of the kidney, while the other, which was more serpentine and longer, crossed the anterior aspect of the corresponding vein and entered the hilus of the kidney. The second (inferior) arose from the primary iliac artery near the terminal portion, descended along the posterior aspect of the kidney, and after a circuit of about 4 cm. entered a deep groove in the parenchyma of the kidney, but terminated finally in the hilus

The left kidney had two veins arising from the hilus. One was very large and about 11 cm. long; the other was quite small, about 7 cm. long and deeper, The two united and emptied into a common trunk about 1 cm. long, which emptied into the common iliac vein.

The right kidney had two veins which were entirely separate. One arose from the hilus of the kidney and was about 15 cm. long. The other right vein was much smaller and shorter. It followed the course of the inferior renal artery and emptied into the external iliac vein at the point where the latter united with the hypogastric.

Niclot and Heuyer[2] report the following:

Soldier died from pneumonia. No kidney signs. Double renal ectopia was found at autopsy. The right kidney was in the small pelvis, on a line with the sacrovertebral angle; the left kidney was at the same angle at its inferior border, and somewhat more vertical. A fibrous band of about 1 cm. in thickness united the two kidneys. The length of this band was 1.5 cm.

The aorta was divided into the right and left iliac artery 10 cm. above the sacral promontory. At this same level the two common iliac veins, united by an anastomotic vein, formed the inferior vena cava. The right kidney was the shape of a triangle at its base, and flattened from in front backward. The hilus was situated anteriorly between the upper and middle portion. Just above the hilus was a lobule about the size of a small egg, with a groove in front. The renal artery, about 8 cm. long, began in the aorta at the level of the two iliac arteries; it was bifurcated at the upper border of the kidney. One of the branches passed to the right of the hilus of the kidney into the lobule mentioned, the other branch penetrated into the kidney on the posterior surface of the lobule. The ureter was about 12 cm. long and descended vertically into the bladder.

The left kidney was flattened anteroposteriorly. The hilus was on the anterior surface between the upper and middle third. There were three left renal arteries. The first arose from the aorta, passed to the rear of the left kidney and then divided into two branches, one entering the posterior surface, the other passing around to the anterior surface near the hilus. The second renal artery arose from the same portion of the aorta as the first and entered the upper posterior surface of the kidney (upper pole). The third renal artery arose like the other two and entered into the kidney at the posterior surface of the inferior pole. There were also three renal veins. The first renal vein left the kidney by the hilus, passing around the left border and following the groove marked by the anterior branch of the renal artery No. 1, passing behind it and entering the left common iliac vein. Renal vein No. 2 left the kidney via the hilus, passed to the right and made a groove on the right side, which, with the groove on the opposite side, divided the kidney into two lobes. This second renal vein formed the upper part of the anastomotic vein spoken of, which united the left common iliac vein to the right common iliac vein. The renal vein No. 3 arose from the lower pole of the kidney from the posterior surface, near the origin of the third renal artery, and formed the inferior part of the anastomotic artery. The ureter was 15 cm. long, descended vertically into the bladder without any curvature.

There was no testicular ectopia. The right kidney weighed 195 grams, the left 210. The suprarenal capsules were in their place.

S. C. Plummer[3] does not cite any cases of bilateral dystopia.

George Strube[4] refers to four cases in the literature. No personal cases of bilateral dystopia of the kidney. He states that the literature contains quite a number of cases

[1] Un cas d'ectopic rénale bilatérale congénitale chez un femme adulte. Arch. ital. de biol., Turin, 1888, ix, 6.

[2] Sur un cas d'ectopie rénale double congénitale. Bull. Soc. anat. de Par., 1905, lxxx, 384.

[3] Dystopic kidney. Surg., Gynec. & Obstet., 1913, xvi, 1.

[4] Über congenitale Lage-und Bildungs anomalien. Virchow's Arch., f. path. Anat., Berl., 1894, cxxxvii, 227.

of double dystopia where the kidneys were fused. He cites 28 bibliographic references and says that Martin St. Ange made autopsy on an infant that had died shortly after birth and found both kidneys in the small pelvis. They were not fused in any way.

R. Boyd:[1]

Female, age 72, died of chronic disease of the brain and lungs. Autopsy showed renal capsules in usual position, on either side of the spine, immediately below the diaphragm. Right kidney situated in the right iliac fossa, below the cæcum, partially concealed by the right ovary, which had a slight peritoneal attachment to it. The renal artery was given off from the right iliac, close to the aorta.

The left kidney was in the pelvis below the psoas muscle, resting on the sacrum and origin of the pyriform muscle. An artery which arose from the aorta at its bifurcation, in the situation of the middle sacral, entered the upper end of the kidney; another larger branch from the internal iliac artery entered the kidney in the usual position. The kidneys, ureters, and bladder were in healthy condition. The vagina was absent, also the uterus and fallopian tubes. The left ovary was diseased, being occupied by a fibrous tumor of an irregular globular shape, connected by a round ligament smaller than that on the right side.

In the discussion of congenital malpositions, Robert Holmes Greene and Harlow Brooks[2] state, on p. 214, that congenital malpositions of one or both kidneys are not uncommonly seen. As a rule they have but little importance clinically, although in certain cases, as was noted by Osler, they may seriously complicate diagnosis. This is particularly true of pelvic kidneys when pregnancy occurs. In a case occurring in the service of the writers, both kidneys were congenitally misplaced in the pelvic cavity; acute nephritis with fatal uræmia followed a twin pregnancy. The woman had passed through a previous single pregnancy without trouble, but in the twin pregnancy the greatly enlarged uterus so compressed one of the misplaced organs as to produce actual strangulation and gangrene with acute nephritis of the other kidney. The condition was not suspected and was discovered only at autopsy.

[1] Malposition of the kidneys; absence of the vagina, uterus, and fallopian tubes; disease of the left ovary. Med.-Chir. Tr., Lond., 1841, xxiv, 187.

[2] Diseases of the Genito-Urinary Organs and the Kidney. Philadelphia and London: 1908, 2d ed., 605.

Ernst Graser[3] states that among 200 cases of congenital misplacement of the kidneys which he found in the literature, he has detailed information as to about 150. In 12 cases both kidneys were displaced downward; in 103 the left kidney 35 times; in one case both kidneys were on the left side; in 4 instances both kidneys were on the right side; in 22 the displaced kidney lay at the level of the promontory; 15 times it was entirely in the small pelvis, usually in the vicinity of the sacro-iliac synchondrosis. He does not state where he found references to the 12 bilateral cases of dystopia which he mentions. He merely says he found 12 cases in the literature.

Potherat and Morderet[4] cite the following:

Female came to autopsy. Both kidneys rested in the lower part of the iliac fossa, along the superior strait of the pelvis. The kidney of the right side is largely destroyed by a cystic degeneration without any macroscopic changes in the ureter. The kidney of the left side is increased in size, probably due to compensatory hypertrophy; the ureter is healthy. The double displacement is congenital for two reasons: (1) The suprarenal capsules occupy their normal position and correspond to the usual kidney distance. (2) The kidneys receive their arteries from many sources. The right kidney receives two arteries of same volume, the upper arising from the aorta immediately below the superior mesenteric and passes behind the vena cava. The inferior artery arises 3 cm. above the iliac bifurcation, at the level of the inferior mesenteric, passing in front of the vena cava. The left kidney received three arteries; one large upper artery arising from the aorta below the superior mesenteric and at a point corresponding to the normal origin of the renal divides into two branches, an upper and lower. The first is subdivided into two rami which enter the upper half of the kidney in front, while the other also subdivided into two branches which pass behind the veins and enter the kidney behind at the hilus.

The second artery, which is also large, arises from the aorta about 1 cm. above the origin of the inferior mesenteric. It passes to the lower end of the kidney, passing behind the corresponding vein, in front of the ureter. The third, very small artery, arises from the aorta at the same level as the superior mesenteric, passes in front of the upper renal artery, in front of the corresponding renal veins, and enters the middle portion of the internal border of the kidney.

[3] Beitrag zur Pathologie und chirurgischen Therapie der Nierenkrankheiten. Deutsch. Arch. f. klin. Med., 1895, lv, 465.

[4] Ectopie congénitale des deux reins. Bull. Soc. anat. de Par., 1889 lxiv, 124.

The patient also presented an anomalous situation of a portion of the duodenum, being located in the inferior portion of the abdomen in front of the superior strait of the pelvis.

P. Poirier[1] examined the cadaver of an old female which showed bilateral renal dystopia.

The right kidney was situated in the posterior half of the iliac fossa of the same side. It had a normal volume. The hilus was in front. There was a large vein 7 cm. long left near the hilus which emptied into the vena cava near or at a point between the second and third vertebræ. No arterial vessel emanated from the hilus. . . . Six arterial branches from various directions entered the kidney parenchyma at different points of the periphery. The first of these branches arose from the aorta about 1 cm. above the bifurcation and emptied into the upper pole of the kidney. The three others arose in a bouquet formed near the angle of the aorta and right common iliac. They entered the posterior portion of the kidney. The fifth arose from the aorta in the angle of the bifurcation of the two iliacs. The sixth which was quite large, arose at the bifurcation of the common iliac. Of all these arteries only one was accompanied by a vein.

The left kidney, much lower than the right, was situated in the pelvic cavity near the median line, about 2 cm. from the body of the fifth lumbar vertebra. Like the other kidney it was slightly lobulated in shape. Its hilus was directly anterior with calyx and one large vein which entered the vena cava at the same point as the left common iliac vein. The kidney received four arteries. The highest arose from the aorta at its bifurcation. The second arose in the same bifurcation angle. The third arose from the bifurcation of the common iliac. The fourth, which had a diameter of 2 mm., arose from a common trunk to the ischium.

The ureters emptied into the bladder normally; the left was 19 cm., the right 24 cm., long. The urinary apparatus, aside from the dislocation of the kidneys, appeared to be entirely normal. The genital apparatus was also normal with the exception of a few cysts in the ovary.

Octave Pasteau[2] reported a case as follows:

Female, age 50, right kidney situated as low down as the right iliac fossa, right lumbar fossa absent. Its inferior extremity was situated 3 cm. below the iliac crest. The kidney was surrounded by a very thick covering of sclerotic tissue which was with difficulty separated from the kidney capsule. The kidney was large, ovoid in shape. It was distended by a purulent fluid, and on incision revealed old pyelonephritic lesions. The right ureter was large, irregular, and filled with pus.

The left kidney was also displaced but situated higher up than the right. Its lower pole extended 2 cm. above the iliac crest. It was not distended. The blood-vessels of both kidneys arose from their normal level, but varied as to their length and course. The general course was downward and backward. The right renal artery was 10 cm. long, the vein 8 cm. The left renal vein from aorta to its opening into the upper border of the kidney measured 8 cm. Three and one half cm. from its origin arose a branch which passed to the lower border of the hilus and measured 5 cm.

He thinks the report of this case of interest because there existed a bilateral dystopia, not congenital in origin, but due to the anomalous origin and length of the vascular pedicle of both kidneys. He found a glandular mass in the pelvis surrounding the right ureter, coinciding with cancer of the cervix uteri.

Max Sträter[3] states that Cadoré collected 74 cases of renal dystopie, most of which were observed at autopsy. Thirteen were right, 29 left, and 3 bilateral ectopias, the location not noted in the remainder. Of the 67 cases collected by Sträter, ectopia occurred 34 times on the left, 19 times on the right, bilaterally in 2 cases, and supernumerary in 2 cases.

Case 1. Reported by Werth (references not given). Female, age 22. Never menstruated; pain in abdomen at intervals of 4 weeks, radiating to back. External genitalia well developed. In place of vagina a blind sac half centimeter deep. Rectal examination failed to reveal vagina, uterus, or adnexa. Instead the finger palpated bilaterally a tumor the size of a fist. Both bodies lay alongside the promontory. The upper end could not be reached. Both bodies were slightly movable. The kidney could not be found in either right or left renal fossa. A diagnosis of double pelvic kidney was made. There also existed a bilateral ovarian hernia, which was removed by operation, by removal of ovaries. Of the tubes only the abdominal end existed; this ended blindly.

Case 2. Elischer (Orvosi hetilap, 1901. Reviewed in *Zentralbl. att für Gynäkologie.*). Did laparotomy on 19 year old girl with defect of vagina, for assumed unilateral hæmatometra. The tumor was recognized, however, to be both kidneys lying in the cul-de-sac of Douglas. The uterus was a rudimentary bicornis.

Says nothing further about bilateral dystopia. Discusses other forms of dystopia in extenso.

[1] Anomalie des reins. Bull. Soc. anat. de Par., 1889, lxiv, 436.

[2] Ectopie rénale double avec pyélo-néphrite droite. Bull. Soc. anat. de Par., 1897, lxxii, 213.

[3] Beiträge zur Pathologie und Therapie der kongenitalen Nieren dystopie. Deutsche Ztschr. f. Chir., 1906, lxxxiii, 55.

Félix-Léon Cadoré[1] in a monograph on congenital anomalies of the kidney cites 3 cases of bilateral ectopia kidney. One by Poirier; one by Potherat and Morderet; and a third case probably by Turner, 1861, of which he says merely:

Case of malformation and malposition of the kidneys; death from Bright's disease; abscess in the kidney and pericarditis.

Under Cruveilhier, 1884, he says as follows: "In a woman two kidneys were fused, both occupying the rear of the rectum, and encroached slightly on the superior strait."

In his incomplete or imperfect bibliography he renders 519 references. Makes no special mention of bilateral dystopia with the exception of the cases mentioned above.

To the above cases, the writer wishes to add the following:

Mr. H. L., 38, married, German, occupation tailor, Richmond, Virginia, referred by Dr. S. B. Moon for investigation of apparent Dietl's crisis from which he had been suffering constantly for several years.

The family history was negative. He had had most of the minor diseases of childhood, but had never contracted typhoid fever, pneumonia, or any severe illness. The bowels were fairly regular; he had gonorrhœa at the age of 25 which ran a mild course, so that he had not noticed anything about the act of urination in recent years which he considered unusual or out of the ordinary.

Examination showed him to be a thin man; height 5 feet, 9 inches; weight 128 pounds; rather pale, with protruding belly and atonic muscles, evident visceroptosis. Heart negative. Lungs showed a slight bronchitis. Liver normal in size. The urinalysis on admission to Grace Hospital was 1,010, alkaline, occasional blood-cells, moderate amount of pus, some large round cells, no casts. Blood count: Leucocytosis of 7,600. Wassermann negative. Luetin test negative. Rectal examination showed a small prostate, and normal vesicles; no tumor could be felt; there were slight hæmorrhoids; no varicocele. Stool: No ova; guiac test negative. No kidney could be felt on the left side, and the right kidney-wall could not be palpated on account of pain and right abdominal rigidity.

On the right side there was a long rectus incision which had been performed about 6 months previously. And in the right lumbar region a tumor could be easily outlined about the size of the palm of the hand, exquisitely tender and apparently the kidney. Investigation of the operation which had been performed upon him October 1, 1913, showed that while in great pain, which had continued for about a week, he had been carried to a hospital where a most competent and efficient surgeon operated, finding a tense empyema of the gall-bladder which was removed, along with an infected, subacute appendix. The dystopic kidney was recognized at the time of this operation and the surgeon attempted extraperitoneally to replace the organ in its normal bed, but on account of a profuse hæmorrhage which started from a large plexus of veins in a centrally located pelvis, nothing could be done and the organ was left untouched. The urinalysis at the time of this operation was amber, clear, acid, no albumin, no sugar, few granular casts.

[1] Les anomalies congénitales du rein chez l'homme. Thésis de doct., Par., 1903, No. 144. p. 205.

Fig. 1. Left pelvic kidney; position below the synchondrosis opposite the second and third sacral foramina; diminutive size; pelvic capacity about 2 ccm.

The patient made a good convalescence and left the hospital in 3 weeks only to have a return of the outbursts from which he had previously suffered, characterized by great pain in the upper abdomen, with occasional vomiting and slight temperature. These attacks persisted throughout the following 18 months with increasing frequency and severity and could only be controlled by large doses of morphine.

We decided that the right kidney was the cause of the outbursts of pain, but there was apparently no left kidney as three X-ray examinations of the left kidney region gave no shadow. On cystoscopy the ureteral meati were somewhat inflamed, the right being the more turgid. Both were normally located, the capacity of the bladder 10 ounces, but the ureteral catheter could be shoved only two inches up the left ureter when it was abruptly stopped.

Pyelography with 35 per cent cargentos showed the left kidney to be at the level of the sacro-iliac notch, its pelvis of so small capacity (about 2 ccm.) that it did not seem it could be of much excretory significance. At this juncture the patient complained of the examination and flatly refused a pyelography of the right side or moth-ball test (see Fig. 1).

The idea then revolved itself around the functionating capacity of the left kidney, which although

Fig. 2. Anterior view of the right dystopic kidney. Note central location of pelvis, flattened condition, and enlargement, dimensions being 5½ x 3¾.

misplaced and diminutive, we hoped would take care of the body so that the right organ, if impossible to anchor in its normal bed, could be removed. It had been planned before operation that a right pyelostomy would be carried out. A phthalein test could then be given, the bladder urine representing the left side, and the pyelostomy drainage that of the right side. If the left excretion proved sufficient, the right kidney could be removed at a later time, as it did not seem justifiable to remove the right organ leaving only the diminutive, ectopic pelvic kidney to serve the body.

Under ether anæsthesia May 19, 1914, an incision was made running from a point below the tip of the ninth costal cartilage downward. The peritoneum was stripped up internally, and was found to be densely adherent to a large plexus of veins, the size of the thumb, in the center of the organ, which was the pelvis of the kidney. A profuse hæmorrhage started when an attempt was made to tease the peritoneum away. The kidney was firmly anchored in its bed and could not be moved. No renal artery was observed, but an aberrant artery at the upper and inner pole about the size of a goose-quill was apparently the only arterial supply the organ enjoyed. Nephrectomy was hurriedly performed on account of uncontrollable hæmorrhage.

The *pathologic condition* of the right kidney as reported by Dr. S. B. Moon, pathologist to Grace Hospital, is herewith given (Fig. 2):

"The right kidney is a symmetric flattened ovoid, its edges laterally placed, its flat surface anterior and posterior. Length 5½ inches, width 3¾inches,

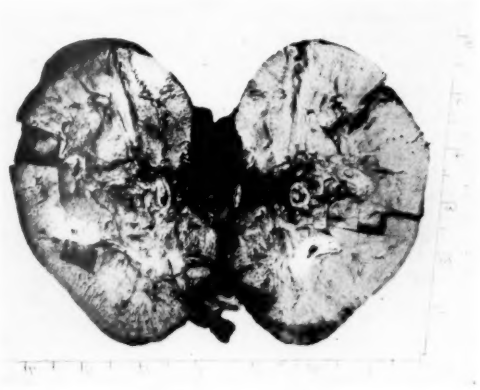

Fig. 3. Opened view, small pelvis communicating with the kidney by three narrow channels; one from the upper pole, one from the lower pole, and one from the middle position. There is no pelvis.

thickness 1½ inches. The surface is smooth save for several slight convex projections, and the hilum is in the middle of the anterior flat surface. An aberrant artery, about ⅛ inch internal diameter, enters the edge one inch internal to the upper pole.

"The ureter, whose wall is decidedly thickened, widens to form a pelvis of proportionally small size, which is pouched communicating with the kidney by only three narrow channels, or calices, one to the upper pole, one to the lower pole, and one to the middle portion.

"The pyramids and cortex are arranged in a more or less haphazard manner, several pyramids, at least, having no communication with the pelvis. The cortex is somewhat thin, the capsule normal, as are also the color and texture, except for several congested areas of inflammation.

"Histologically the kidney structure appears to have been normal both as to medulla and cortex. Some areas exhibit an inflammatory condition with round-cell infiltration and cellular degeneration and desquamation. These areas are especially noticeable at the corticomedullary junction, around the blood-vessels" (Fig. 3).

It was an interesting feature in the consideration of this ptosed right kidney, whether the patient's sartorial occupation predisposed to the abdominal outbursts by the crouching abdominal flexion of his character of work, bringing on a pelvic glaucoma; or whether the dense adhesions to the peritoneum anteriorly restricted and choked the ureter, for he had noticed for several years past that rapid walking, lifting, or straining aggravated the outburst.

Following the operation the patient reacted slowly, 24½ ounces of urine being excreted in the succeeding 24 hours, which was 1,020 acid, no blood, no albumin, small amount of pus, a great many uric acid crystals, no casts, a few large round cells. Remarks: Less pus than before operation.

Urinalysis June 11, 1914: Acid, 1,008, no albumin, greatly diminished phosphates, excessive amount of indican, occasional red blood-cells, large amount of pus; no crystals, no casts, few cells from bladder.

June 12, 1914, the patient left the hospital having had an uneventful convalescence, freedom from his attacks of pain, and declaring that he felt better than he had in several years.

May 1, 1915. The patient has been at work since the fall, has occasional attacks of abdominal discomfort, but none of the severe outbursts which he formerly had. He thinks that attacks of indigestion bring on the pain. He drinks freely of water and voids about every two hours by day, getting up twice at night, something that he never had to do before the operation. The urinalysis shows acid 1,020, no albumin, no sugar, no blood, pus, or crystals, no casts, no excessive mucus. The patient takes no opiate and has increased somewhat in weight.

With the development of pyelography, X-ray, cystoscopy, and ureteral catheterization, distorted states of the genito-urinary system are more frequently observed now than in former years, an anomalous state of the kidney being observed about once in every 25 cases, for anomalous conditions of the genito-urinary system are not uncommon; they occur more frequently than in any other structure of the body.

Nor is this hard to explain when we bear in mind the complex embryological development of the urogenital system:

Etiology. In early life the cloaca is both the future rectum and bladder. A vertical fold separates the bladder anteriorly from the rectum posteriorly. The wolffian duct gains the cloaca dorsally and ultimately forms the renal pelvis and ureter. The kidney slowly ascends to the lumbar region, in the process of fœtal evolution and development, receiving in its excursion arterial twigs which are given off from successively higher points on the aorta; as the kidney reaches higher, these twigs atrophy, drop off, and new arteries supplant them. The artery which continues as the renal artery is the last stem claimed by the kidney.

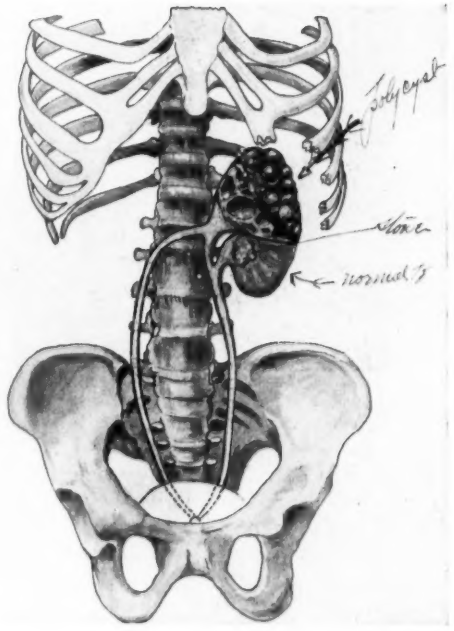

Fig. 4. An anomalous right kidney (no kidney found on the left side) with the right ureter coming from the upper half of the pelvis, the upper pelvis communicating with the lower pelvis and left ureter, a stone present in the pelvis with the upper two-thirds of the kidney polycystic and the lower third normal kidney tissue. J. W. Vander Veer's case.

In anomalous dystopic states, the arterial feed to the kidney may come from along the course of the aorta, of from any of its subdivisions on the corresponding side, from the bony pelvis below to the diaphragm above. The case of transplantation of the kidney in the anterior abdominal wall is but an extreme exaggeration of its possible congenital dystopia, the organ taking the anterior instead of the normal posterior ascent. If, in its forced excursion upward, violating gravity, visceral influences, growth, and arterial traction, one stem refuses to give up its nutritive right, the kidney is tethered, and with development and embryological growth the artery thickens and becomes permanent, fascial increase is noted, and the kidney is anchored in moorings which nature has not intended.

Pathology. That anomalous kidneys, the fused, *nieren-kuchen* sigmoid, or horseshoe

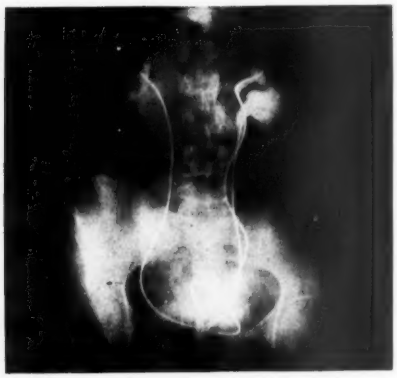

Fig. 5. Three kidneys and three pelves. The lower one on right hand side had numerous tubercle bacilli.

kidney should be invaded by some pathological state and is particularly liable to disease and a morbid reversion, has been well established and appears to be constantly found. The case of Dr. J. W. VanderVeer well illustrates this (Fig. 4):

"An anomalous right kidney (no kidney found on the left side) with the right ureter coming from the upper half of the pelvis, the upper pelvis communicating with the lower pelvis and left ureter, a stone present in the pelvis with the upper two-thirds of the kidney polycystic and the lower third normal kidney tissue."

In cryptorchismus and hernia of the ovary that these organs tend to malignancy is well known. This malignant predisposition seems to obtain also in the acquired ectopic kidney. An anomalous illustration is that mentioned by Retcke in 1898.

He reports in extensive detail a case of dislocation of both kidneys as the result of a fall from a wagon wheel to the ground.

The patient struck his back upon the hub of the wheel and the wagon tongue and from there he fell to the ground striking firmly on the buttocks. He noted a severe pain in the back at the time and became unconscious for 10 or 15 minutes. Subsequent developments showed traumatic right wandering kidney. The patient declined operative fixation and left the hospital much improved June 30, 1894. In January, 1897, he developed chills and fever, and pain in the abdomen and back, similar to the symptoms he showed at the time of dislocation of the right kidney, only this time the pain was on the left side. In the interim the right mobile kidney had also given him trouble. Both kidneys were displaced downward to a considerable extent. The condition of the patient grew worse so rapidly that malignant disease of the left kidney was suspected. Surgical measures were again refused. Death October 29, 1897.

The writer is convinced that malignant disease of the left kidney had developed but autopsy was also refused.

A résumé of the necropsy findings in 18 authentic cases of bilateral ectopy shows the following constant development inhibition:

1. Kidney fixed, densely bound down, flattened.
2. Pelvis usually situated anteriorly.
3. Located about sacro-iliac synchondrosis; *i.e.*, below the pelvic brim.
4. One small single artery, may be as many as six twigs, entering the organ at an abnormal site.
5. Veins multiple, much enlarged, emerging at an angle.
6. Adrenal bodies in their normal subdiaphragmatic locations.
7. Lobulation of the kidney.
8. Hypoplasia of calyces.
9. Ureters are standard but shorter.

Associated with ectopic kidney, any genital anomaly may be observed as epispadias, and hypospadias, extrophy, bicornate uterus, cryptorchismus, atresia of the anus, absence of vagina, absence of one ureteral meatus, etc.

The writer has reported the case of three ureters and three pelves, two on the right side, the lower one of which had developed a tubercular invasion, with hæmaturia and urinary distress (Fig. 5).

Most cases of renal dystopia, however, are recognized on the table in the course of an abdominal operation, the condition having been unsuspected.

We are limiting our discussion to, and considering only the congenital dystopic kidney and not the wandering kidney regardless of its degree of movability, for the mere fact that it moves differentiates the two, for the congenital ectopic kidney whether in the pelvis or abdominal cavity, is bound down by fascial bands and enjoys no excursion.

Differential diagnosis. Strube states that the literature contains quite a number of

cases of double dystopia where the kidneys are fused, and gives 28 bibliographic references. In this condition, the hypertrophied meatus, the excessive peristalsis, capacious, confluent pelves on pyelography and mesial location, should differentiate the horseshoe ectopy from bilateral dystopia — which is relatively much rarer.

The condition seems to be equally divided between the sexes, and it is at the average age of 30 that surgical comfort has been sought.

Subjective symptoms are famously unsatisfactory. Rovsing, however, calls particular attention to pain in the abdomen, lumbar region, and beneath the epigastrium. This pain radiates downward and is relieved by prolonged rest in bed. There is also throbbing and a sense of pressure which is aggravated by leaning backward.

Neufville reports a case in which there was compression of the abdominal vessels and thrombosis of the iliac and femoral veins causing œdema of the legs and ascites.

Green and Brooks refer to a case in which a woman gave birth, both of her kidneys being in the pelvis, at a later day with twin pregnancy, the greatly enlarged uterus causing compression strangulation of the kidneys and gangrene. The condition was recognized at autopsy.

A routine abdominal examination is particularly indicated when renal dystopia is suspected on account of genital anomalies.

The possibility of ectopic kidney, unilateral or bilateral, should be borne in mind in all tumors about the brim of the pelvis and particularly in those instances when the kidney cannot be felt in its normal bed.

Rectal examination should be thoroughly carried out.

The phenolsulphonephthalein test is not of peculiar diagnostic value; X-ray shadows of soft tissues in the abdominal cavity are notoriously misleading.

Urinalysis is not significant and conclusive, for urinary findings and cellular elements may obtain in any renal state.

Morbid states of the ectopic kidney and stone inclusion confound rather than simplify the diagnosis. The embarrassment to neighboring organs is as well misinterpreted.

Cystoscopy may reveal the meati normal or in an abnormal location, hypertrophied and showing excessive peristaltic waves, the catheter being arrested at the location of the tumor. In cases of cystitis and contracted bladder, indigocarmine may be used to locate the ureteral ora.

It is by ureteral catheterization, injection of colloid silver, and X-ray that a constant and reliable means for positive diagnosis can be brought out; for pyelography decides not only the location and capacity of the pelvis, but anatomically differentiates the tumor from a hydronephrotic sac, a revelation which the metal stilette could not accomplish.

Treatment. Treatment is that of the arising surgical indication. If observed in the course of a laparotomy performed for some other separate pathological state, nothing should be done. It is with the state of pregnancy and pressure constriction that radical surgical intervention must be adopted, the unoffending kidney balanced, and if not found wanting, the aberrant organ removed. It would appear that this congenital anomaly, borne for many years, must undergo a distinct pathological reversion to bring out a train of symptoms which would be referable only to the urogenital system. On these occasions routine examination will determine for the surgeon the indications, method, and character of operation.

CORRECTION OF DEPRESSED FRACTURES OF THE NOSE BY TRANSPLANT OF CARTILAGE [1]

E. H. BECKMAN, M.D., F.A.C.S., ROCHESTER, MINNESOTA
From the Mayo Clinic

A LARGE number of nasal deformities resulting from injury are of a depressed type, that is, the injury has crushed the nasal bones so that the bridge of the nose is flattened. The nasal bones are either forced backward upon themselves or separated and pushed out laterally in such a manner that the bridge of the nose is broadened as well as depressed. Many of these, if treated immediately, can be replaced and held with splints. The intranasal splint made of hard rubber and the external splint composed of sheet lead are at times both exceedingly valuable. If such a fracture has been allowed to heal without the bones being replaced a marked deformity often exists. A very slight deformity of the bone may produce marked disfigurement of the entire face.

Various methods of correcting these deformities have been tried. One of the commonest has been the transplantation of a portion of bone from the anterior portion of the tibia. A great deal has been written within the past few years in regard to the transplantation of bone, fascia, and fat. Scarcely any mention has been made in the literature in regard to the transplantation of cartilage. Adult cartilage can be transplanted from one portion of the body to another under the same conditions that bone is transplanted, with uniformly good results. Transplantation of cartilage differs from the transplantation of bone, because it is not necessary for the viability of the transplant that the cartilage should be in contact with other cartilage or bone. We have also found that it is not at all necessary to preserve the perichondrium in order to secure a good result. Experimental work by Macewen in the transplantation of embryonic cartilage shows that young cartilage when transplanted into the subcutaneous tissues continues to grow, and in this way a great many cartilaginous tumors have been produced in animals. Davis' work shows that when adult cartilage is transplanted it lives with no apparent growth or change in its composition, up to three months.

We have several cases in which adult cartilage has been transplanted from the rib into

Fig. 1, *a*. Before operation.

Fig. 1, *b*. Before operation.

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Fig. 2, *a*. After operation.

Fig. 2, *b*. After operation.

the nose for the purpose of replacing a defect in the tissues there. Some of these cases have gone two and one-half years with no apparent change in the size of the transplant. The tissue accepts the transplant very kindly and even in one case of marked infection the cartilage finally healed in firmly. It is not necessary to transplant cartilage immediately. In one instance a portion of cartilage had been removed from the sternal end of the rib for the purpose of transplanting into the nose, but on account of the congested condition of the patient from the anæsthetic, pneumonia being feared, the cartilage was placed in salt solution and confined in a refrigerator for five days. At this time the patient was again anæsthetized, the cartilage removed from the salt solution, and transplanted into the nose. Primary union occurred just as if fresh cartilage had been used.

TECHNIQUE OF OPERATION

If the nasal bones are widely separated it is best to refracture them and place a lateral splint upon each side of the nose, letting the splint remain for five or six days, in order to procure a suitable narrowing of the bridge of the nose. As soon as it is certain that there are no breaks in the nasal mucous membrane and that the circulation about the nose is again perfect, the transplant can be made. I have followed the custom of taking a portion of the costal cartilage from the seventh rib, since the cartilage is much broader at this point and a good sized portion can be obtained, still leaving costal attachment between the ribs and sternum. One is often surprised to find that a large portion of cartilage is necessary to remedy an apparently small defect. A wax model of the nasal defect is valuable as a guide in shaping the cartilage which may be easily whittled with a scalpel to any desired size or shape. No attention need be paid to the perichondrium.

An incision a quarter of an inch in length is made transversely through the skin over the nose and just between the two inner canthi at a point where the bridge of the ordinary spectacles rest. There is usually a slight wrinkle in the skin at this point so the scar is not visible. With a periosteal elevator the skin and subcutaneous tissues are elevated from the bone and cartilage straight down the bridge of the nose nearly to the tip. The separation should not be carried laterally on either side farther than necessary to secure room to transplant the cartilage. The transplant is then slipped into place, and, as the tissues have been separated only in the midline, the transplant is held in place and is not dislodged to either side. It may be necessary, however, to place a stitch at the upper portion of the transplant to keep it from slipping upward. The small incision

Fig. 3. Before operation.

Fig. 4. After operation.

is closed with two or three interrupted stitches of horsehair; the wound sealed with cotton and compound tincture of benzoin.

In my first attempt to transplant cartilage into the nose an incision at the rim of the nostril was made, the cartilage inserted at this point. This method has also been recommended by Babcock. It is not so satisfactory as the method herein described because the dissection cannot be kept exactly in the midline and consequently the transplanted cartilage is apt to slip sidewise. The risk of infection is also much greater in the former method. The only infection in our cases was the one in which a transplant was placed in through the nostril. The accompanying photographs illustrate the points in the technique and the results obtained.

SOME INTERESTING EXPERIENCES IN THYROID SURGERY[1]

By ALEXIUS McGLANNAN, M.D., Baltimore

THE INFLUENCE OF CHRONIC TONSILLITIS ON EXOPHTHALMIC GOITER

INCREASED activity of the thyroid gland is one of the many factors involved in our resistance to infection. Enlargement of the gland is so constantly associated with the course of an acute infection that it seems a normal function. The intensification of the symptoms of hyperthyroidism whenever an infection is engrafted on a previously existing exophthalmic goiter points out the close relation existing between the thyroid gland and the reaction of the organism to injury.

This relationship seems particularly marked when the infection exists in the throat or the accessory sinuses of the nose. For some time we have had our thyroid patients studied

[1] Read at the Annual Meeting of the Medical and Chirurgical Faculty of Maryland.

Fig. 1. Photograph[1] of the patient in Case 1 (Sur. 5,105, St. Agnes' Hospital) before operation.

by the rhinologist and any lesion of the nose or throat treated before making the direct attack on the thyroid.

The following case illustrates the value of this proceeding:

CASE 1. Surgical No. 5,105, St. Agnes' Hospital. White male; age 23; occupation farmer. The onset occurred five months before admission. The initial symptoms were: nervousness with tremor of the hands, sleeplessness, and some bulging of the eyes. Later he noticed enlargement of the neck, intermittent attacks of diarrhœa and progressive loss of weight — twenty pounds in four months. The patient gave a history of nasal catarrh lasting 13 years, an acute suppuration of the ear occurring 4 years ago, and an attack of quinsy 14 months before admission.

On admission he presented the typical picture of exophthalmic goiter (Fig. 1, showing appearance of patient in Case 1): large goiter with distended veins and purring thrill; tachycardia with enlarged area of cardiac dullness; marked exophthalmos with practically all the special eye signs; profuse sweating, gastric hyperacidity; leucocytes, 6,680, small mononuclears, 26.8 per cent; total lymphocytes, 36.8 per cent; urine negative. X-ray examination did not show an eniarged thymus. Examination of the throat revealed large infected tonsils, the crypts of which contained cheesy exudate.

[1] Photographs for Figs. 1, 2, 3, 4, 5, and 6, by Mr. H. Schapiro. Pathological specimens from the Surgical Pathological Laboratories of the Johns Hopkins Medical School and the College of Physicians and Surgeons, Baltimore.

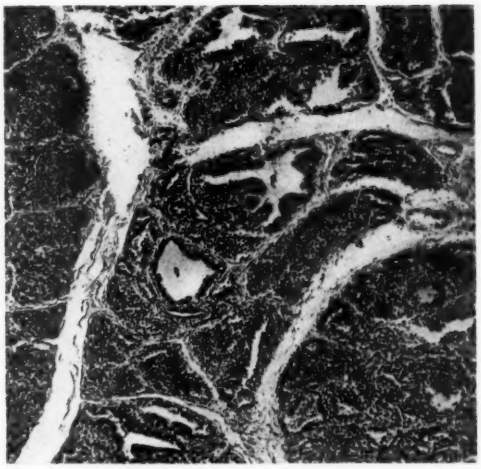

Fig. 2. Photomicrograph of section of thyroid from Case 1 (Path. 16,455, exophthalmic goiter).

After rest in bed for 12 days, the pulse rate had fallen from 140 to 100. On this date Dr. Pound removed the left tonsil under novocaine anæsthesia. The immediate reaction was quite severe, as is shown by the anæsthesia slip record. After the operation the pulse rate rapidly fell, and at the end of 24 hours it was 110, and after 48 hours 100.

Two weeks later the second tonsil was removed. The operation was performed with considerably less disturbance, the pulse and blood pressure gradually falling after the initial excitement had subsided. The post-operative tachycardia was rather persistent, the pulse rate averaging about 120 for ten days. After this time the rate was usually from 100 to 120, averaging 112.

Two weeks after this tonsil was removed, that is, 39 days after admission and 27 days after the first tonsillectomy, I removed the right lobe of the thyroid under novocaine and drop ether anæsthesia. During the operation the pulse rate averaged 130 and the blood pressure 160. The anæsthesia slip shows how slightly the patient was disturbed by the operative manipulations. During the first 24 hours after operation the pulse rate varied between 130 and 150, and during the next 24 hours came down step by step to 110, reaching a range between 80 and 100 on the fourth day after operation.

Fig. 3. Photograph of the patient in Case 2 (S. 2,849, Mercy Hospital).

Pathological examination of removed thyroid shows advanced exophthalmic hypertrophy (Fig. 2). The cut surface has the appearance of muscle with some areas of colloid remaining in the gland.

The beneficial effects of the tonsillectomy in this patient are, first, the diminution in the intensity of the hyperthyroidism after the removal of the tonsils, which permitted the very satisfactory and safe removal of the thyroid lobe; second, the absence of severe reaction after the lobectomy, which was no doubt due to the preliminary unburdening of the system by means of the tonsillectomies. It is most unusual to have so prompt a recovery with so little intoxication following operation for so well-marked an exophthalmic goiter.

Fig. 4. Photograph of lobe removed from patient in Case 2 (Path. 1,746, simple goiter).

LARGE SIMPLE GOITER WITH HYPOTHYROIDISM

CASE 2. Surgical No. 2,849, Mercy Hospital. White female; age 37; the mother of six children,

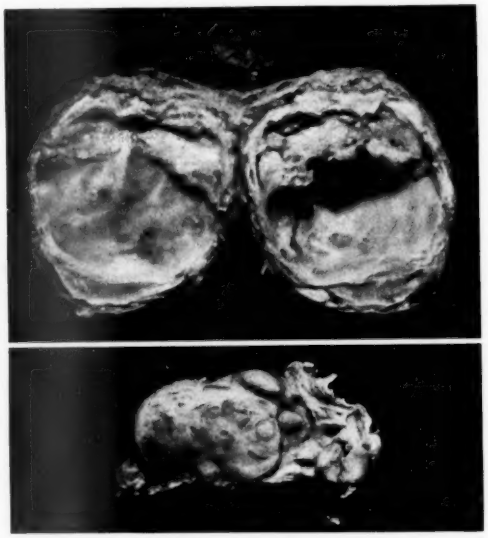

Fig. 5 (above). Photograph of cyst with ossification of the walls, removed from patient in Case 3 (S. 1,363, Mercy Hospital).

Fig. 6. Photograph of the mass removed from patient in Case 4 (Path. 904, aberrant thyroid).

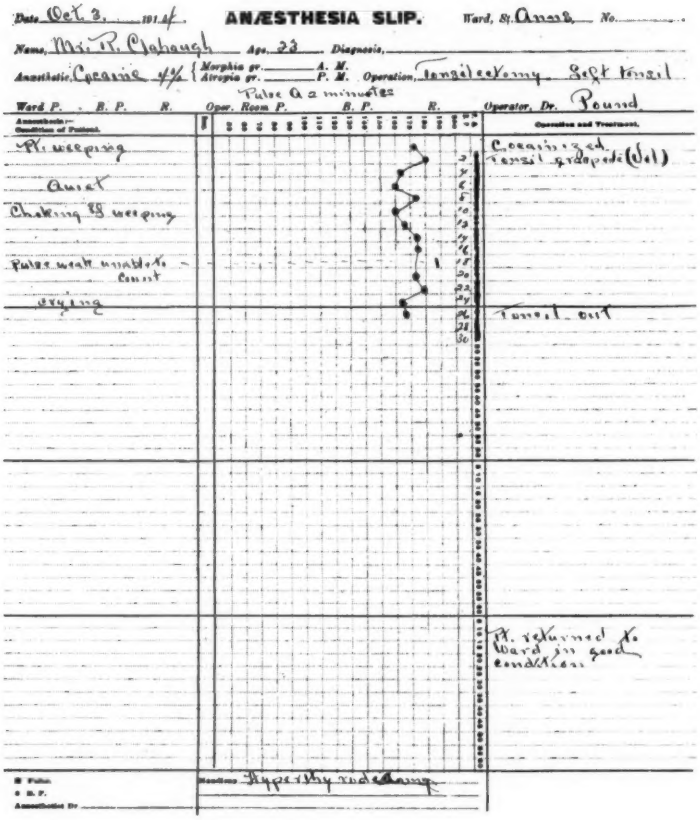

Fig. 7.

Figs. 7, 8, and 9. Anæsthesia slips showing pulse rate and blood pressure during successive tonsillectomies and thyroidectomy.

the youngest being 4 years old. The onset was 13 years ago, with birth of first child. The initial symptom was a swollen thyroid. The swelling increased with each pregnancy. During the past six years the patient has become quite fat and has gained 90 pounds in weight. For past three years she has suffered from dyspnœa and hoarseness.

The patient is quite fat, of short stature, and the body is held in the kyphotic position. (Fig. 3 shows appearance of patient in Case 2.) Hair coarse, considerable growth of hair on chin and upper lip. The menstrual history is negative. The mental processes are somewhat slowed. The skin all over the body feels thick and is thrown into folds at the position of all the ordinary creases. Both lobes of the thyroid are enlarged, extending from well up in the submaxillary region down under the clavicle. The goiter is smooth and does not pulsate. There is marked dyspnœa and hoarseness, with a little difficulty in swallowing. The pulse rate is 64, blood pressure 130.

The patient was kept in bed and given thyroid extract. The kidney function (phthalein test) was 29, with some albumin but no casts in the urine. On forced water the kidney function gradually improved until it reached 42 and remained at about this point. Dried pituitary was given for several days, but without any noticeable effect.

After twenty-two days of preliminary treatment, operation was performed under novocaine and ether anæsthesia. The left lobe, which extended from the level of the hyoid bone to a distance two fingers' breadth below the clavicle, was first removed. Until this lobe was dislocated no ether could be administered on account of the dyspnœa. About one-third of the right lobe was next removed and the wound closed.

Since operation the patient's general condition

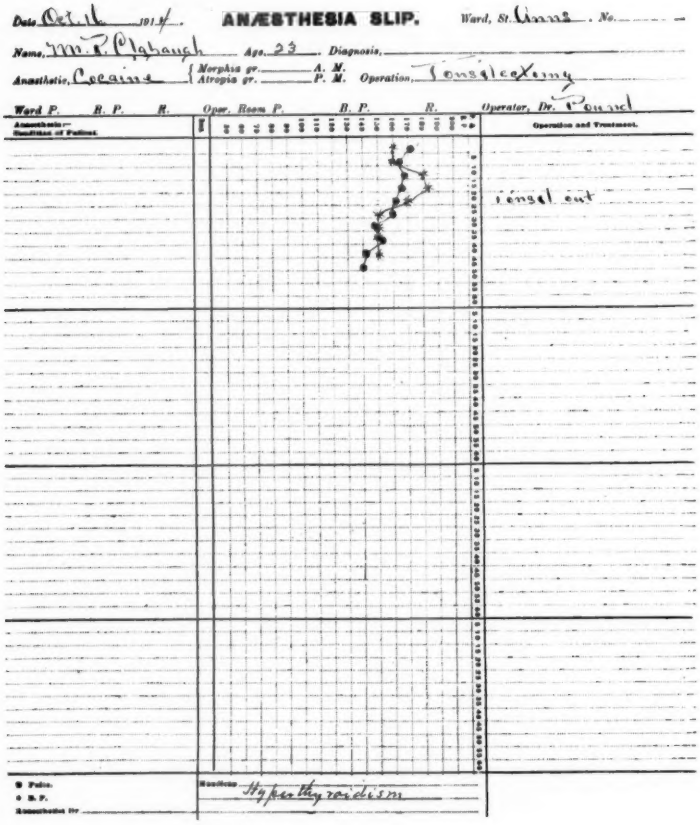

Fig. 8.

has steadily improved, and her mental condition has become quite acute.

The tissue removed is a simple goiter, showing the large vesicles, with hæmorrhagic areas and connective-tissue proliferation in the stroma (Fig. 4).

HYPOTHYROIDISM ASSOCIATED WITH THE PRESENCE OF A LARGE OSSIFIED CYST IN THE THYROID

CASE 3. Surgical No. 1,363, Mercy Hospital. White male; age 35. The duration of thyroid lesion was unknown. The patient was admitted to the hospital on account of a scaly eczematous eruption covering the entire body. The thyroid mass was discovered in the routine physical examination. The pulse rate averaged about 70; leucocytes, 6,000; small mononuclears, 23 per cent; total lymphocytes, 25 per cent. The thyroid mass situated in the left lobe was very hard and smooth of outline.

The association of the thyroid mass with the skin lesion and the blood picture noted indicates one of the incomplete forms of hypothyroidism. Accordingly the patient was given thyroid extract by mouth. The skin lesion gradually healed, and after thirty days' treatment had disappeared. At this time the proportion of small mononuclears was 25 per cent; total lymphocytes, 32 per cent.

A few days later operation on the thyroid was performed under ether anæsthesia. The mass proved to be a cyst, with ossification of its wall, and was situated in the lobe. A thin layer of thyroid was turned back with the capsule, and the stone-like body was separated from the glandular tissue by blunt dissection. The thyroid tissue was pale, almost white, and had an abundant fibrous stroma.

The tumor seemed to be the result of ossification of a thyroid cyst. We have had cysts whose walls were partly calcified and

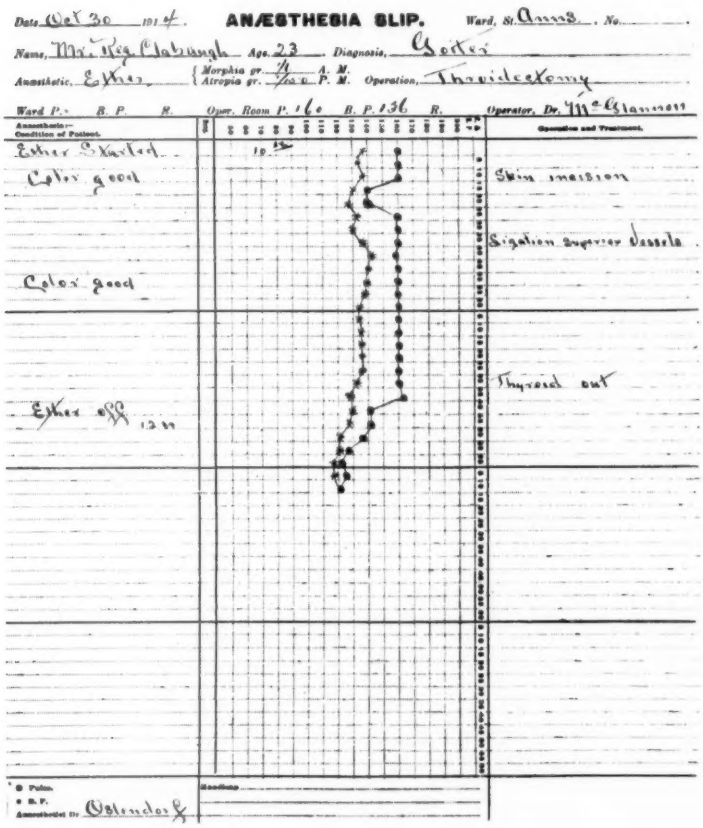

Fig. 9.

have had areas of calcification and of bone in simple goiters and thyroid adenomas, but this is the largest calcified mass in our collection (Fig. 5).

After the operation the patient recovered without any complications, and although no more thyroid extract was administered his skin remained healthy. We hope that the improved circulation in the left lobe that must follow the relief of pressure brought about by removal of this mass will so add to its function as to meet the demands of his body.

ADENOMA OF LATERAL ABERRENT THYROID

CASE 4. Surgical No. 228, Mercy Hospital. White male; age 27. Patient has had a lump in the posterior triangle of the left side of his neck for three years. This has gradually become larger and lately has become tender. On examination a number of enlarged glands were found on both sides of the neck, both anteriorly and posteriorly. The glands of the groin also were found enlarged. The mass noticed by the patient was larger than any of the swollen glands and was the only tender one. There was no fever; leucocytes, 5,400; small mononuclears, 20 per cent; total lymphocytes, 26 per cent. The patient did not react to a tuberculin injection.

The pre-operative diagnosis was glandular enlargement due to lues or to Hodgkin's disease. When the mass was exposed in the wound and the characteristic greenish-red vascular capsule came into view, the thyroid tumor was recognized and removed.

Pathological examination of the tumor showed the usual picture of colloid adenoma of the thyroid with some thyroiditis.

THE RÔLE OF OXYURIS VERMICULARIS IN THE ETIOLOGY OF APPENDICITIS AND ALLIED PATHOLOGICAL CONDITIONS[1]

By KWANNOSUKE SUZUKI, M.D.
Fleet Surgeon of the Imperial Japanese Navy

ALTHOUGH the above subject has long occupied the attention of numerous writers, this problem has not yet received its solution. The opinions touching it are very varied, some workers considering the presence of the oxyuris in appendicitis to be purely accidental, while others attribute great pathological significance to the presence of but one such organism. Nothing would be gained by reviewing the existing literature on this subject; I will therefore confine myself to a few brief comments on one or two important contributions to this subject, and then pass on to an exposition of my own findings.

The point of first importance is that which during the past few years has served as the basis of the controversy existing between Rheindorf and Aschoff, and which has not yet reached its solution. As is known, Aschoff[2] formerly maintained that the parasites were unable to play an important etiologic rôle in appendicitis. At that time out of a thousand appendices examined by him, of which the greater number were pathologically affected, only two cases were found by him to exhibit the presence of oxyuris. On the other hand, Rheindorf[3] has since 1912 published several papers on the subject of the oxyuris as a factor in appendicitis, in which he declares that this parasite plays an important rôle in the etiology of the disease, especially in the case of children, and that it is far from being the harmless parasite it was formerly held to be. In his first work, Rheindorf published the result of the examination of thirteen appendices, all derived from children from three to twelve years old, who had appendicitis. Six of these cases showed the oxyures in the appendix (47.6 per cent). His microscopic illustrations show the presence of a deeply fissured ulceration at the site where the organisms were found in the mucous membrane. The bed of the ulcer, together with various ramifications of the same, extends practically to the musculature. At the edges of the same a moderate leucocytic infiltration is visible.

Aschoff[4] later on changed his opinion with respect to the matter, and in part confirmed the statement of Rheindorf by declaring that the pathologic condition provoked was not true appendicitis but a diseased condition similar to the latter, caused by the oxyures, which condition he termed "appendicopathia oxyurica." The point most hotly contested by these two authors is whether the oxyures can provoke a fissured and canalicular formation in the mucous membrane. Rheindorf affirms that this is the case, while Aschoff most persistently denies its possibility, and even insists that this formation is merely an artefact.

In 1913 Rheindorf[5] published a fresh contribution to the subject, entering exhaustively into a description of the pathologico-anatomical changes brought about in the wall of the appendix by the parasite. At the same time he declared most emphatically that the cleft in the mucous membrane could undoubtedly be produced by the oxyures, and was by no means an artefact.

Recently Rheindorf[6] has issued an additional contribution, reaffirming his last statements, in which he further explains that the cleft formed by the oxyuris is to be differentiated from another due to an artefact. But Aschoff[7] replied to this by de-

[1] Aschoff. Die Wurmfortsatzentzündung, 1908, Jena.

[2] Rheindorf. Beiträge zur Frage der Bedeutung der Oxyuren bei der Wurmfortsatzentzündung der Kinder. Berl. klin. Wchnschr., 1912, Nos. 10 and 11, 451, 503.

[4] Aschoff. Appendicopathia oxyurica, Med. Klin., 1913, ix, No. 17, 249.

[5] Rheindorf. Über die durch die Oxyuris vermicularis hervorgerufenen pathologisch-anatomischen Veränderungen in der Wand des Wurmfortsatzes. Frankfurter Ztschr. f. Path., 1913, xiv, No. 2, 212.

[6] Hysteroneurasthenie oder chronische Appendicitis? Zugleich ein Beitrag zur Appendicitisfrage und ihrer Beziehung zur Oxyuris. Berl. klin. Wchnschr., 1914, Nos. 27 and 28, 1211, 1271.

[7] Aschoff. Sind die Würmer, besonders die Oxyuren, direkt oder indirekt schuld an der Appendicitis? Berl. klin. Wchnschr., 1914, No. 32, 1504.

[1] This work was conducted from October, 1913, until August, 1914, in the Pathologic Institute of the Städt. Krankenhaus, Urban, Berlin, under the direction of Dr. Max Koch, Director of the Institute. I here wish to express my sincere thanks to Dr. Koch for his friendly assistance in the work.

claring that the oxyuris clefts of Rheindorf were purely artificial in origin. Nevertheless, he confirmed some of the other statements of Rheindorf. Among other remarks we find the following: "(2) The description made by me several years ago of the pathologic picture of pseudo-appendicitis, due to the presence of oxyuris, has been acknowledged by Rheindorf and confirmed in its essential points. (3) The fissure formations in the mucous membrane of the appendix, described by Rheindorf and attributed by him to the entrance of the oxyures, are for the most part artificially produced. (4) So long as we have no proof that the oxyures are able to provoke coarse epithelial defects or tissue destruction, we are similarly unable to accept Rheindorf's statement that the oxyures are indirectly connected with acute appendicitis."

In addition to the two authors quoted above, various other investigators have offered contributions to the solution of the problem. Hippius and Lewinson[1] in their earlier writings have also expressed the opinion that traumatic lesions of the wall of the appendix are caused by the oxyures, and that infectious agents are thus able to penetrate into the tissue of the appendix. This opinion is theoretically quite correct, although inflammation is not always caused by the presence of the oxyures in the appendix, even in cases where the organisms penetrate deep into the mucosa, and sometimes also into the lymph-nodules as far as the proximity of the submucosa or the musculature, where they cause traumatic injury to the wall of the appendix.

Latterly N. Sagredo has examined 100 appendices which were extirpated in connection with appendicitis, among which he found 41 cases showing the parasites or their eggs. These 41 appendices, with the exception of 6 cases, showed no important pathological changes. On the other hand, appendices exhibiting intense inflammation very rarely show the presence of the parasite. The 35 cases, therefore, which were operated upon for appendicitis-like symptoms, must through the agency of the oxyures have provoked a clinical picture identical, not with that of appendicitis, but, to use Aschoff's designation, with that of "appendicopathia."

It appears to us erroneous to attempt to explain this question merely from a consideration of the statistical data, without undergoing a thorough investigation of the pathologico-anatomical changes in the appendix wall; for, on the one hand, the appendix can harbor the oxyures without any accompanying abnormal clinical symptoms, and, secondly, the patient may be suffering from acute pain in the region of the cæcum, even in the total absence of the parasite. From Sagredo's article we are left uninformed as to the whereabouts of the oxyures in the appendix; and furthermore what is the nature of the anatomic changes (other than the inflammatory process) brought about by the nematode, and what is their connection with the clinical symptoms? According to our own investigations the anatomical changes of the appendix accompanying the presence of the oxyures are not only inflammatory, but traumatic disturbances, localized necrosis, and intense hyperplasia of the lymphatic tissue, etc.

Hueck[2] examined 78 resected appendices, amongst which he found 14 cases of oxyuris in the lumen of the appendix, and one case of the organism in the submucosa. He made microscopical examinations of cross-sections from at least three different places in each of the appendices (usually proximal, medial, and distal), and summed up his investigations in the following words: "This points to the fact that in many cases often designated as appendicitis, the agent responsible for the infection is the nematode. According to my own experience the oxyures alone are to be held responsible for the specific complications of the appendix. Thus, we are concerned not with appendicitis, viz., an inflammatory reaction, but with a diseased pain-producing condition of the appendix, which may be termed "appendicopathia oxyurica."

The same author found no cases in which true inflammation was caused by the oxyures, or in which the wall of the appendix had been

[1] Hippius and Lewinson. Oxyuris and Appendix. Deutsche med. Wchnschr., 1907, xxxiii, No. 43, 1780.

[2] Hueck. Über die pathologische Bedeutung von Helminthen in der Appendix. Frankfurter Ztschr. f. Path., 1913, xiii, No. 3, 434.

traumatically injured. He confined his observations to resected appendices, and, unlike Sagredo, studied no normal cases.

Matsuoka[1] recently examined 103 appendices, and a part of his findings have already been communicated by Aschoff. Matsuoka found 33 cases of recent (histologically confirmed) appendicitis (9 per cent), and 26 cases showing the oxyuris from among 70 cases without any recent inflammation (37 per cent). For a detailed account of his findings we must await the publication of his entire work.

I should here like to emphasize the importance, in the study of oxyuris appendicitis, of examining not only resected appendices, but also normal appendices derived from post-mortem material. In my own study of post-mortem cases I found several cases of localized oxyures, unaccompanied by any apparent anatomic changes or other clinical symptoms.

At the instigation of Dr. Max Koch, I have endeavored to cast some light on this problem. The main points which have received attention are the following:

1. What are the pathologico-anatomical changes produced in the appendix through the agency of the oxyures, and are these anatomical changes to be regarded as being artefacts?

2. Can the inflammation, whether acute or chronic, be caused by the oxyures?

3. Can a disease of the appendix clinically similar to appendicitis be provoked by the oxyures without any accompanying inflammation?

4. Can the last-named disease of the appendix arise in the wall of the processus without any apparent anatomic changes?

5. What is the percentage of the occurrence of inflammatory or other pathological conditions in connection with the oxyures?

For the purpose of solving the above questions I examined 500 post-mortem cases, among which were 44 oxyuris cases. The appendices of these 44 cases were cut into serial sections and subjected to careful microscopic examination. In addition to this post-mortem material examinations were made of 103 appendices which had been extirpated from patients suffering from appendicitis.[2] Serial sections were made from the material taken from children under 17 years of age, and sections of the appendices obtained from the patients above the age of 18 were cut in serial sections, and subjected to careful microscopic examination at intervals of from five to ten sections.

[1] Aschoff. Sind die Würmer, besonders die Oxyuren, direkt oder indirekt Schuld an der Appendicitis? Berl. klin. Wchnschr., 1914, No. 32, 1504.

PREPARATORY STUDIES

As a preparation for my investigations I made a histologic comparison of the species oxyuris and trichocephalus. These two nematodes are somewhat similar in aspect when examined microscopically, especially when seen in a cross section of their anterior end, although macroscopically they are very unlike, and can be distinguished at a glance. Specimens of these nematodes taken from post-mortem cases were fixed in alcohol-sublimate (1:4), embedded in paraffin in the usual manner, cut 5 microns thick in various directions, stained with hemalum-eosin, and examined microscopically (Figs. 1 and 2). I wish to say that the use of the hemalum-eosin stain is very much to be recommended in the examination of these parasites, for the cuticula consisting of a chitinous substance stains intensely with eosin and acquires a clear and shining aspect, whereas the viscera for the most part, as well as the yolk in the egg, take on the hemalum stain. The egg-shell in the case of both species remains unstained, but is sharply outlined, reflecting the light. I made various other attempts at staining, but failed to obtain equally satisfactory results.

The fact that the cuticula takes on the eosin stain so intensely greatly facilitates the search for the oxyures, and thanks to this method, we are easily able to detect the presence in the tissue of the most minute portion of a worm. In cross sections of the posterior ends, the two organisms can be easily distinguished by the difference in the anatomic position of the viscera; also by the mas-

[2] This material was obtained from the operating room of Prof. Körte, Director of the Chirurg. Abt. d. Städt. Krankenhaus at Urban, Berlin. I here wish to express to Prof. Körte my sincere appreciation of his generous aid.

sive cuticula in the neighborhood of the organs possessed by the trichocephalus, as well as by the form of the eggs. But in a cross section of the anterior end of the body the two species are often seen to bear close resemblance to one another. It is especially difficult to determine the species when in a section of the anterior end a fragment of the cuticula and of the pharynx or œsophagus is seen. I have, however, discovered a satisfactory means of differentiating the two types; for under a strong magnification the cuticula of the trichocephalus is seen to consist of two layers, an outer colorless layer and an inner layer stained with eosin, whereas in the oxyuris the cuticula consists of but one layer, sharply stained with eosin (Figs. 3 and 4). In transverse sections of the anterior part of the body, I have found yet another important point of differentiation. This consists in the fact that whereas in trichocephalus the musculature of the pharynx and œsophagus consists of a dotted side-by-side disposition of the small bundles of muscles, in oxyuris on the other hand it is seen as a long, uniform, and uninterrupted band of muscle.

As has been remarked, the eggs of these two nematodes, when seen in their entirety, vary greatly in respect to their general shape, and can be differentiated at a glance. But in my investigations I was not able always to easily recognize an isolated egg which happened to be situated in the lumen or in the tissue, and this was especially the case where the sections of the eggs of the trichocephalus were neither directly transverse nor vertical, and where, as a consequence, the characteristic covering at the two poles was not present. In such cases there remains but one manner of differentiation, and that is the fact that in the oxyuris the yolk takes the hemalum stain fairly intensely, whereas the trichocephalus almost entirely fails to do so; moreover that in oxyuris a broad intervening space is present between the egg-shell and the yolk, whereas in trichocephalus no such space is seen (Figs. 5 and 6). It is sometimes very important to discover the isolated unattached eggs which may be situated in the lumen or tissue, for one is thereby enabled to assume that the appendix is infected with the nematodes. In the tissue we sometimes find an empty egg-shell; but even then the two species can be distinguished by the fact that the egg-shell of oxyuris reflects light more sharply and that its contour is more distinctly outlined.

A transverse examination of the appendix is to be recommended in the majority of cases, as such a procedure best demonstrates the various relations which may exist between the processus and the nematodes. It is generally known to be exceedingly hard to point out the exact situations of the worm in relation to the appendix-wall when the organism is situated longitudinally in a fold of the mucosa, and examined in a longitudinal section, because the portion of the parasite which appears in such a section does not indicate whether it is actually lying within the fold or outside of the same. Longitudinal sections were prepared only occasionally, when necessary.

The histologic differentiation between male and female specimens of the oxyuris can only be made in longitudinal sections or in transverse sections through the part of the body containing the genital organs. In the female the genital tract is usually completely filled up with eggs (Figs. 7 and 8). Apart from this point, the sex of the nematodes cannot be made out histologically.

If we wish to institute a detailed investigation of the connection of the oxyuris in appendicitis, we must examine the whole appendix by means of continuous serial sections, or at least one out of every 5 or 10 sections throughout the entire processus, although this involves a great amount of labor. Hueck made microscopical examinations of only three transverse sections taken from different sites (usually proximal, medial, and distal), but I hold this procedure to be entirely too crude.

EXAMINATION OF POST-MORTEM MATERIAL

I examined 500 cadavers, finding the oxyures in 44 cases, as follows: 29 times (66 per cent) in the cæcum and appendix, 6 times (14 per cent) in the cæcum alone, and 9 times (20 per cent) in the appendix alone.

The following table shows the results with respect to oxyuris of the examination of the 500 post-mortem cases.

Age in Years	Number of Cadavers Examined			Total Number of Oxyures Found			Number of Trichocephalus Found	Proglotides Tapeworm[1]
	Male	Female	Total	Male	Female	Total		
1	13	8	21	0	0	0	1	
2 to 10	14	8	22	10 71 per cent	3 38 per cent	13 59 per cent	2	
11 to 20	17	11	28	4 29 per cent	1 9 per cent	5 18 per cent	2	1
21 to 30	25	18	43	2 8 per cent	0	2 5 per cent	3	
31 to 40	48	18	66	5 11 per cent	1 6 per cent	6 9 per cent		2
41 to 50	47	18	65	3 6 per cent	0	3 5 per cent	1	
51 to 60	60	22	82	6 10 per cent	1 5 per cent	7 9 per cent	1	1
61 to 70	67	23	100	3 4 per cent	1 3 per cent	4 4 per cent		
71 to 80	33	29	62	4 12 per cent	0	4 7 per cent		
81 to 90	6	5	11	0	0	0		1
	330	170	500	33 10 per cent	11 6.5 per cent	44 8.8 per cent	10 (2 per cent)	5 (1 per cent)

[1] Only in the appendix.

The various authors are unanimous in affirming that the percentage of cases in which the oxyuris is found is much greater in children than in adults. Out of 11 cases of children who failed to harbor the organism in the cæcum, when examined by means of selective serial sections, 6 showed its presence in the appendix.

Age in years	Number of appendices examined	Number of cases showing oxyures
3	6	2
2	1	1
6	1	1
7	1	1
9	1	1
14	1	0
	11	6 (55 per cent)

The examinations showed that when the consistency of the fæces in the cæcum was hard, it contained more oxyures than when the fæces was liquid. In the latter cases the parasites were usually found in the lower part of the colon.

I examined all the appendices showing oxyures in the cæcum (in serial sections) under the microscope, fixed with formalin *in toto* without opening, embedded in paraffin, then stained with alum-hæmatin-eosin. The result of my examination is as follows:

Specimens of oxyuris, found only in the lumen		29
Specimens of oxyuris found in lumen and in	gland-tube	3
	deep gland-tubules	2
	mucosa	3
	lymph-nodes	2
No specimens of oxyuris in appendix		6
Specimens of trichocephalus found in lumen		6
Tape-worm proglotides, found in lumen		5[1]

[1] Already found microscopically.

1. *Oxyures found in the lumen.* Sometimes the oxyures are found in the lumen of the appendix (the mass of fæces being of varying consistency), situated in part between the fæces and the wall of the processus; at other times they are found in the lumen alone, sometimes surrounded with mucus. Occasionally only a few eggs, either isolated or in groups, are seen in the lumen, distributed among the fæces, without any parts of the parasites themselves being present (Fig. 9). The origin of these eggs may be accounted for in two ways: First, they may have entered the lumen of the appendix either through traumatic bursting of the female or through its deformation brought about by any other means. Second, they may have emerged from the body of the female when the latter was in the cæcum, and thence have proceeded to enter the lumen of the processus, together with the fæces. Nevertheless, I am inclined to the opinion that when an egg is found in a

group it will have left the body of the mother while the latter was in the lumen of the appendix. Occasionally a free, isolated egg is found in the tissue (Fig. 10). Rheindorf holds that the fertilized oxyuris eggs are deposited in the lumen of the appendix, and that they there develop into the embryonic organism, and finally into the adult. Some years ago Küchenmeister represented the view that the oxyures were able to develop in the cæcum and appendix beside the adults, without the necessity of their previously passing through the mouth and stomach of the host. Heller and others believe this opinion to be erroneous. Hueck stated that it was hard to say with certainty that they had been liberated through the deformation of the female. Michelson[1] and Vignolo-Lutati[2] had earlier proved that the eggs can continue to develop on the skin.

In order to elucidate this point I instituted a number of experiments. Two dogs were fed by mouth with a number of oxyures bearing fertilized eggs, and in two other dogs the impregnated females were introduced directly into the cæcum. My object was to ascertain whether or not the oxyuris could develop in the intestine to the adult stage without undergoing a passage through the outer world. But contrary to expectation the oxyures failed to develop in all four experiments. Of course this experiment must be pursued still further in order to decide the question; but so long as we have no proof that the oxyuris eggs are deposited in the intestine and there develop into the adult form, I am inclined to think that Rheindorf's theory is erroneous. At all events I myself have never seen in the appendix cases of embryonic organisms which had recently left the egg. The above-stated findings, on the contrary, incline me to the belief that eggs found in a large group, whether in the appendix, the lumen, or the tissue, must have been forced out of the body of the mother through some special cause, but that only eggs found free and isolated can be taken as having been deposited naturally.

The formation of depressions in the mucous membrane, corresponding to the position of the oxyures is, according to my mind, merely mechanical in origin, and is produced by the pressure of the worms on the mucosa. These depressions, moreover, are most pronounced when the oxyures are situated between the hard fæcal mass and the mucosa (Fig. 11). On the other hand, when they remain quite loosely in the lumen, where there is no fæcal matter, no depression whatever is noted (Fig. 12). This depression is sometimes seen to be deeply indented, in which case the oxyuris will penetrate the same, and it will appear at a glance as if the worm had bored through the mucosa (Fig. 13). Examination with a high-power microscope, however, shows that no traumatic disturbance of the tissue is involved, that the edge of the adjoining mucous membrane is uniformly smooth, and that the cells at this place are sometimes greatly compressed. The epithelium, however, is often entirely absent at this place or very much injured, and at times extensive maceration of the epithelium is to be noted.

Rheindorf thought that the destruction caused to the epithelium by the oxyures was not to be attributed solely to the purely mechanical effect of the movements of the anterior and posterior ends of the body, but that a contributory factor might be a certain chemical noxa given off by the parasite, resulting in the destruction of the epithelium. My own observations, however, do not lead me to support this theory of Rheindorf, as neither defects nor maceration of the epithelium were to be seen in cases where the oxyures failed to exercise pressure on the mucosa; or, in other words, where no mechanical effect was produced by the action of the worm on the mucosa. In cases where the oxyures in the lumen are not in such close contact with the mucosa, the epithelium invariably remains intact. If it were really destroyed by some chemical action, the epithelium in the neighborhood of the oxyuris would always have to be injured, even though no mechanical action had here taken place. I had no difficulty in explaining these conditions. Figure 14 shows some sections of oxyures in the lumen together with some mucosa, and one specimen of the organism is even seen to be quite close to the mucosa. But the epithelium in its

[1] Berl. klin. Wchnschr., 1877, No. 33.
[2] Arch. f. Dermat., lxxxvii.

entire circumference is well preserved. In another figure we have a transverse section of two oxyures in the lumen, surrounded by liquid fæces; here, however, we see neither destruction nor maceration of the epithelium (Fig. 15). In another case the greater part of the epithelium is absent at the site of the location of the oxyures; the edge of the mucosa, nevertheless, is perfectly smooth, and no tissue destruction has taken place.

Rheindorf has further stated that between the ulcerated mucous membrane and the oxyures could be seen an agglomeration of leucocytes, lymphocytes, and detritus, with a possible admixture of the fæces of the parasite. This statement is correct, for we do see such a mass of detritus in the lumen of the appendix. But it is conceivable that this mass of detritus is on the one hand a physiological result of the metabolism of the host, and secondly, a result of the long-continued mechanical irritation produced by the worms or by the hard fæcal matter. My own preparations often failed to show any such formation of detritus, but merely a certain amount of mucus, although the oxyures were in contact with the mucosa.

My own observations, therefore, induce me to state that the destructive effect of the oxyures on the epithelium is only to be attributed to a mechanical cause and that we can hardly entertain the theory of the liberation by the parasite of a chemical noxa. Rheindorf even holds that a chemical noxa capable of causing epithelial destruction is given off by the isolated egg; but even this hypothesis is unacceptable, as the existence of the isolated eggs is not always associated with tissue decomposition.

Rheindorf attributes great pathological importance to the destruction of the epithelium of the mucosa. My own investigations have shown that the epithelium in post-mortem cases is indeed greatly disturbed, even when no oxyures are found in the appendix or any other pathological changes are seen to occur. The material extirpated from the appendicitis patients, on the other hand, usually shows a well-preserved epithelium, even when oxyures are seen to be present and even when severe histological lesions are found. These findings incline one to the belief that part of the epithelial destruction found in the post-mortem material is to be considered as due to post-mortem decomposition. The material obtained from operated cases was fixed immediately after extirpation, while yet warm; but post-mortem material was fixed later, after death. In children's cadavers 10 per cent formalin solution was sometimes injected into the abdominal cavity as soon after death as possible, in order to prevent post-mortem decomposition. It should be emphasized that all epithelial destruction found in post-mortem cases must be examined with the utmost care and should be carefully distinguished from post-mortem changes.

2. *Oxyures in the gland-tube.* Two specimens of oxyuris were found in which the head had penetrated the gland-tube, while the body had for the most part remained in the lumen. One of the accompanying illustrations shows an oblong section of the anterior body of the oxyuris which has entered a gland-tube, the epithelium of which has been completely destroyed by the worm where it is in contact with the organism. The wall of the gland-tube was quite smooth, and no tissue destruction here took place. In another illustration (Figs. 18 and 19) we see that the oxyuris head has half penetrated the gland-tube, and in the space around the head of the organism a few detached epithelial cells can be seen. Both these cases failed to show either leucocytic infiltration or necrosis of the tissues in the neighborhood of the oxyuris. The rest of the appendix was completely normal.

In one of the sections I found a particle of oxyuris cuticula lying in a deeply-seated gland-tube, the epithelium of which had been completely destroyed, but the wall of which was as smooth as in the previously quoted cases. This gland-tube is situated in the vicinity of the muscularis mucosæ. Here likewise there was no leucocytic infiltration, nor were there any noteworthy histological changes in the tissue, the entire wall remaining intact (Figs. 20 and 21). In another figure (Fig. 22) we see a minute scale of oxyuris cuticula lying in the gland-tube on the surface of the mucosa; the lumen shows one

Fig. 1.

Fig. 2.

Fig. 3.

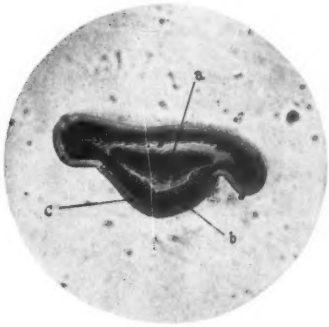

Fig. 4.

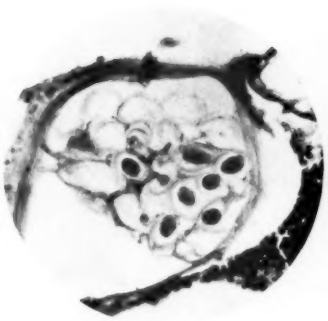

Fig. 5.

Fig. 6.

Fig. 7.

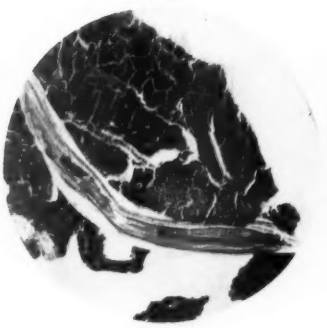

Fig. 8.

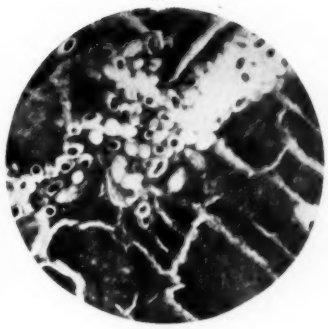

Fig. 9.

Figs. 1 to 32. Post-mortem material. Fig. 1. *a* and *a'*, Transverse section of the anterior body of oxyuris vermicularis; *b*, longitudinal section of the anterior body; *c*, transverse section of the posterior body; *d*, small fragment of the cuticula of oxyuris vermicularis.

Fig. 2. *a*, Transverse section of head end of trichocephalus dispar; *b*, transverse section of posterior end; *c*, longitudinal section of anterior body.

Fig. 3. Greater magnification of transverse section of head end of Fig. 1, *a*. *a*, Œsophagus. *b*, cuticula consisting of one single layer, intensively stained with eosin.

Fig. 4. Greater magnification of transverse section of trichocephalus, posterior end, from Fig. 2, *a*. *a*, Œsophagus; *b*, inner layer of the cuticula, intensively stained with eosin; *c*, outer layer of cuticula, unstained.

Fig. 5. Oxyuris eggs in the uterus, possibly fertilized; coarser yolk, intensively stained with alum-hæmatin.

Fig. 6. Trichocephalus eggs in the uterus. *a*, Plugs at both poles; *b*, fine yolk, stained with alum-hæmatin; *c*, eggs at the plug.

Fig. 7. Longitudinal section through the middle of a female oxyuris, filled with eggs.

Fig. 8. Longitudinal section through middle of male oxyuris, at site of digestive tract and seminal canal.

Fig. 9. Group of free oxyuris eggs, scattered in the hard fæcal matter in the lumen of the appendix.

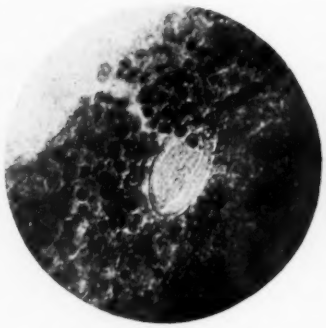

Fig. 10.

Fig. 11.

Fig. 12.

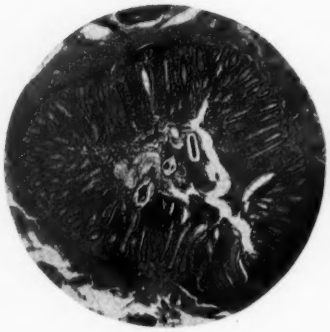

Fig. 13.

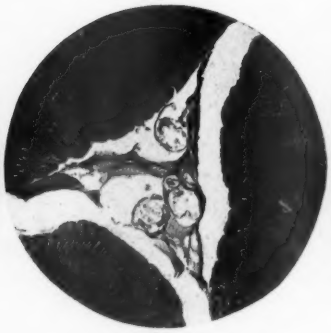

Fig. 14.

Fig. 15.

Fig. 16.

Fig. 17.

Fig. 18.

Fig. 10. An isolated, free oxyuris egg in the tissue of the mucosa.

Fig. 11. Various oxyuris specimens in the lumen of the appendix. Arrow shows depression formation.

Fig. 12. Two specimens of oxyuris in the lumen of the appendix. *a*, Depression formation; *b*, enveloped in mucus, no depression formation.

Fig. 13. Various oxyuris specimens in the lumen of the appendix; arrow shows mucosa compressed by oxyures.

Fig. 14. Four transverse sections of oxyuris found in the lumen of the appendix, accompanied by some mucus; oxyuris situated quite close to the mucosa. Epithelium well preserved.

Fig. 15. Two specimens of oxyuris situated between the liquid fæces and the wall of the appendix. Epithelium well preserved.

Fig. 16. Five specimens of oxyuris in the lumen of the appendix, one of which is entering a gland-tube (arrow).

Fig. 17. Greater magnification of the above preparation. *a*, Longitudinal section of the anterior body in the gland-tube; *b*, smooth wall of the gland-tube; the epithelium is absent at the part adjoining the oxyuris; *c*, well preserved epithelium at the bed of the gland-tube.

Fig. 18. Three specimens of oxyuris in the lumen, one of which is in the act of entering the gland-tube with its head.

Fig. 19.

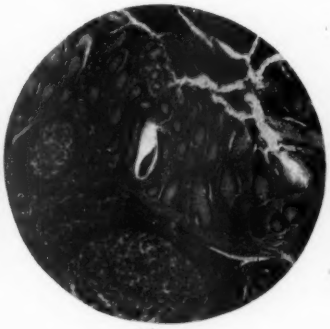

Fig. 20.

Fig. 21.

Fig. 22.

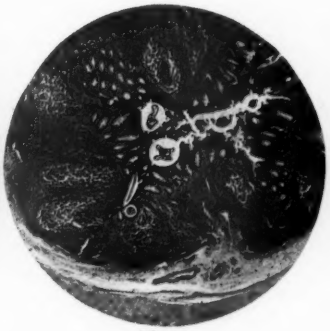

Fig. 23.

Fig. 24.

Fig. 25.

Fig. 26.

Fig. 27.

Fig. 19. Greater magnification of previous figure. The head end reaches to the middle of the gland-tube, where several fallen-out epithelial cells are visible.

Fig. 20. A fragment of oxyuris cuticula at the end of a gland-tube; several epithelial cells are missing.

Fig. 21. Greater magnification of the preceding figure. Smooth wall of the gland-tube; epithelium is entirely absent.

Fig. 22. Two specimens of oxyuris in the lumen between the fæces and the mucosa; one is seen transversely and the other longitudinally; there is likewise a small transverse section in the orifice of the gland-tube (see arrow).

Fig. 23. Two specimens of oxyuris in the lumen, a longitudinal section of oxyuris in one gland-tube, and a transverse section in another.

Fig. 24. Greater magnification of the previous figure (arrow). Several remnants of nuclei of the epithelium of the gland-tube; sharply outlined, smooth wall where the epithelium is entirely absent.

Fig. 25. An oxyuris in act of penetrating mucosa.

Figs. 26 to 29. Series showing the condition of the penetration of oxyures in the mucosa.

Fig. 26. Penetrating mucosa, between two gland-tubes.

Fig. 27. Further penetration; a gland-tube pressed to one side.

Fig. 28.

Fig. 29.

Fig. 30.

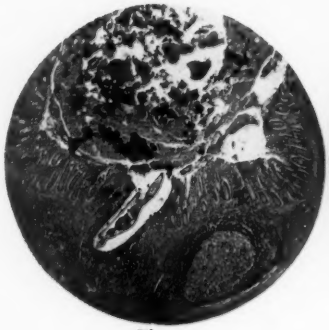

Fig. 31.

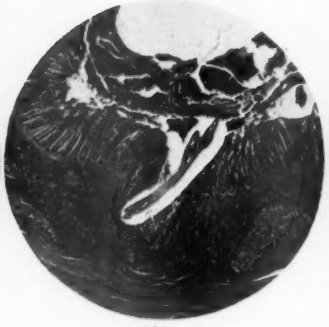

Fig. 32.

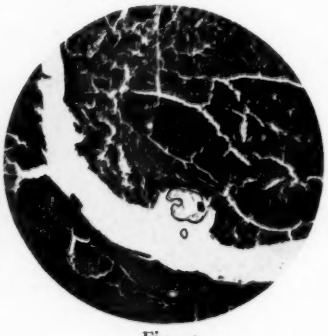

Fig. 33.

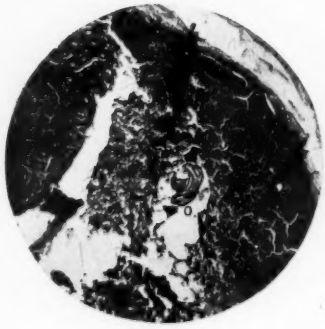

Fig. 34.

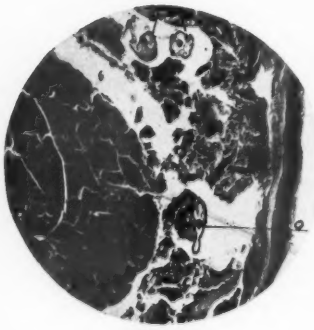

Fig. 35.

Fig. 36.

Fig. 28. Penetrating under the epithelium.

Fig. 29. Oxyures situated directly under the epithelium; the epithelium well preserved.

Fig. 30. Two transverse sections of oxyures in the lymph-nodules, situated quite close to the submucosa.

Fig. 31. Oxyuris boring into the mucosa from the lumen; the wall of the fissure is quite smooth.

Fig. 32. Series following the preceding figure. The oxyuris has bored through the muscularis mucosæ and has reached the lymph-nodules; the wall of the fissure is quite smooth.

Fig. 33. Chronic appendicitis; lumen filled with hard fæces. An oxyuris between the fæces and the mucosa. Arrow shows artificially formed cleft, caused by fixation.

Fig. 34. Chronic ulcerous appendicitis with extensive destruction of the wall, lumen, and defects of the wall, which is filled with liquid fæces; an oxyuris is seen in the defect, as well as some hard fæcal matter.

Fig. 35. Chronic ulcerated appendicitis. Lumen is filled with hard fæces, and the wall has been destroyed as far as the muscularis. Three oxyuris specimens are visible in the defective wall.

Fig. 36. Acute suppurative appendicitis with perforation. *a*, Perforation; *b*, two oxyuris specimens in the suppurated infiltrated lymphatic tissue.

Fig. 37.

Fig. 38.

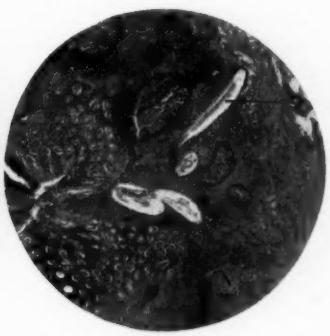

Fig. 39.

Fig. 40.

Fig. 41.

Fig. 42.

Fig. 37. "Appendicitis oxyurica." This shows a transverse section of the proximal end of a wall. *a*, Oxyuris in the lumen; *b*, two oxyuris sections in the mucosa; *c*, scleronecrotic part; *d*, necrosis with hæmorrhage.

Fig. 38. Inflamed mucosa and submucosa. Transverse section of the middle part of the wall. Several oxyures in the lumen, embedded in the fæces, several oxyures in the mucosa and submucosa; necrotic destruction of tissue accompanied by hæmorrhage in the vicinity of the oxyures. The entire epithelium is destroyed, acute inflamed submucosa (*sb*); circular muscle (*cm*) and long muscles (*lm*).

Fig. 39. Transverse section of distal end of wall. Four specimens of oxyuris in the mucosa, one of which extends to the submucosa; several slightly necrosed foci with hæmorrhage in the inflamed mucosa.

Fig. 40. Transverse section of middle of wall. Torn mucosa; several oxyures in the lumen, four oxyuris sections in the lymph-nodules accompanied by necrosis and hæmorrhage.

Fig. 41. Transverse section of the wall at the top of the distal end, where the lumen is no more found. One oxyuris in transverse section; *a*, necrosis; *b*, hæmorrhage; *c*, acute inflamed lymphatic tissue.

Fig. 42. Greater magnification of Fig. 38. *a*, Torn mucosa; *b*, necrosis and hæmorrhage in the neighborhood of the oxyures; *c*, the fæcal mass between the oxyures in the mucosa.

longitudinal and one transverse section of the oxyures.

A further case examined showed the worms in the gland-tubes. This is seen as a longitudinal section of an oxyuris in a longitudinally cut gland-tube, as well as cross sections of both. Around the cross sections no more epithelium is to be seen, and the glandular wall is consequently quite smooth, while along the longitudinal sections of the gland-tube we nevertheless still see a few remnants of the epithelial nuclei. This case showed no noteworthy histological changes in the tissue, with the possible exception of a certain compression of the cells surrounding the glands (Figs. 23 and 24).

In the two cases first described it is easy to explain the destruction of the epithelium by the purely mechanical action of the oxyures, since only the epithelial cells directly in contact with the oxyures suffered destruction, while those situated in the vicinity of the parasites, but not adjoining the same, remained intact. If this effect were produced

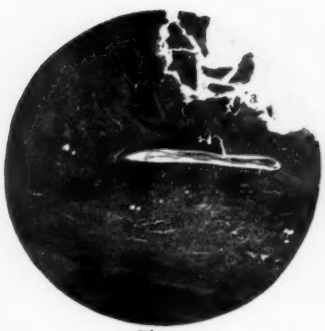

Fig. 43.

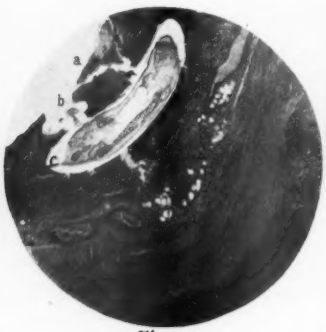

Fig. 44.

Fig. 45.

Fig. 46.

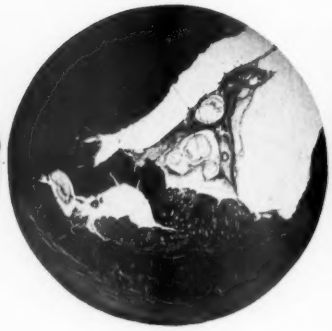

Fig. 47.

Fig. 48.

Fig. 43. *a*, Torn mucosa; *b*, an oxyuris longitudinal section in the mucosa; greatly inflamed submucosa (*sb*), circular muscles (*cm*) and longitudinal muscles (*lm*).

Fig. 44. *a*, Destroyed mucosa; *b*, oxyuris passage, which is connected with the lumen and contains a small fragment of oxyuris; *c*, a large longitudinal section of a female oxyuris in the lymphatic tissue; in the neighborhood of the oxyuris there is necrosis and hæmorrhage. Severe acutely inflamed submucosa (*s*). Circular muscles (*cm*) and longitudinal muscles (*lm*).

Fig. 45. Greatly destroyed mucosa in the vicinity of the lymph-nodules. *o*, Oxyuris section in the cleft of the tissue.

Fig. 46. "Appendicopathia oxyurica traumatica." The epithelium is well preserved, showing no inflammation. *o*, Several oxyures in the lumen; *o'*, oxyuris in the mucosa; *g*, oxyuris passage; *r*, a large previously formed space, produced by a large diverticulum-like indentation of the wall; partly destroyed by oxyures.

Fig. 47. Continuation of series. The epithelium is well preserved, and no inflammation has occurred. *o*, Oxyuris in the lumen; *o'*, oxyuris in the mucosa; this oxyuris fissure communicates with the large space by means of a passage.

Figs. 48 to 52. "Appendicopathia oxyurica traumatica." The epithelium is well preserved, but partly destroyed; there is no inflammation. *o*, Oxyures in the lumen; o^1, oxyuris in the act of penetrating into the mucosa; o^2, o^3, penetrating still further; o^4, the last position of the oxyuris in the series; *h*, hæmorrhage in the lumen; *h'*, hæmorrhage in the tissue surrounding the oxyuris.

by a chemical noxa liberated by the parasite, as Rheindorf suspects, the epithelium of an entire gland-tube should in these cases have been affected (see Fig. 17).

3. *Oxyures which have penetrated the wall of the appendix.* The opinions of the various authors respecting the localization of the oxyures in the wall of the appendix are very different; indeed, it is even a matter of controversy whether the oxyures are able to penetrate an intestinal wall that is entirely normal. Since Wagener[1] in 1905 first demonstrated the presence of the oxyuris in the intestinal wall, this problem has been variously explained by different authors. Schneider[2] had previously described a dead female oxyuris found in the pelvic peritoneum of a woman; but he was of the opinion that it had reached this site by way of the fallopian tubes,

[1] Wagener. Virchows Arch. f. Anat. u. path. Anat., 1905, clxxxii, 145.

[2] Schneider. Oxyuris vermicularis im Beckenperitoneum eingekapselt. Zentralbl. f. Bakteriol. u. Parasitenk., 1904, xxxvi, 550.

Fig. 49.

Fig. 50.

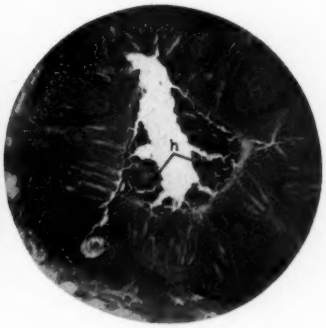

Fig. 51.

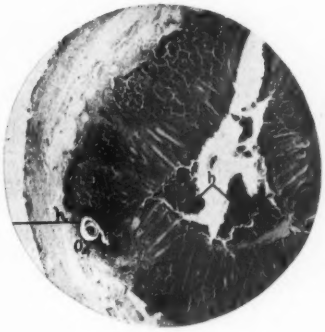

Fig. 52.

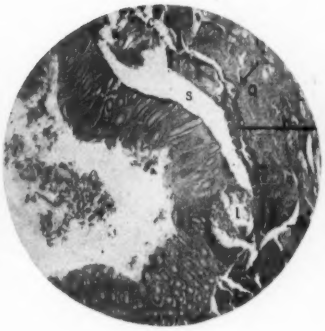

Fig. 53.

Fig. 54.

Fig. 55.

Fig. 56.

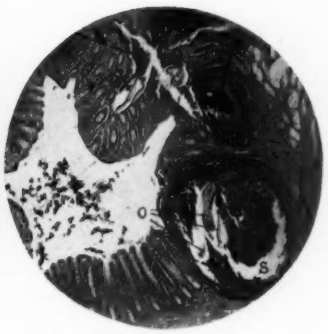

Fig. 57.

Figs. 53 to 64. "Appendicopathia oxyurica traumatica," with pronounced fissure formations and lymph-nodule destructions.

Fig. 53. *o*, A small oxyuris section; *s*, a large fissure; *c*, torn mucosa; *h*, hæmorrhage in the fissure; *l*, destroyed lymph-nodules; *s'*, a cleft to be considered an artefact, which may, however, have been formed during the process of fixation. It is situated at the edge of the lymphatic tissue.

Fig. 54. Next in series Figs. 53 to 59.

Fig. 55. Same series. *o*, Oxyuris in an extensive fissure with hæmorrhage in the lymphatic tissue, whose position corresponds to the arrow in the preceding illustration. Oxyuris fissure without any oxyures. At the same place in the next series an oxyuris is seen.

Fig. 56. Same series. *o*, Oxyures, *s*, oxyuris cleft, *h*, blood in the fissure, *h'*, hæmorrhage in the tissue.

Figs. 57 to 59. Same series.

Fig. 58.

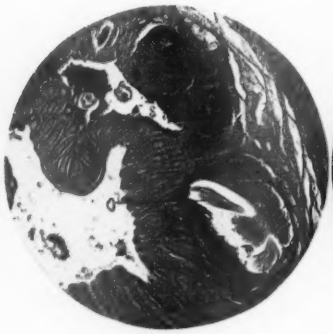

Fig. 59.

Fig. 60.

Fig. 61.

Fig. 62.

Fig. 63.

Fig. 60. *o*, Head end of an oxyuris, which has bored through a lymph-nodule.

Fig. 61. Continuation of the series. *o*, Oxyuris in the center of a lymph-nodule; *s*, destruction of the lymphatic tissue, brought about by the oxyures; *s'*, fissure to be considered an artefact.

Fig. 62 and 63. Continuation of the series showing large defect in lymph-nodule.

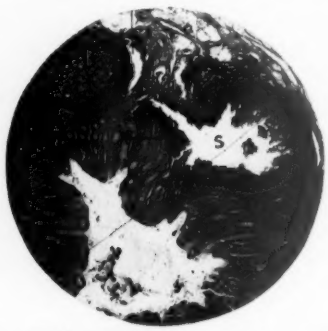

Fig. 64. The last in the series. No oxyures are to be seen. This fissure, nevertheless, represents destruction of the lymph-nodules effected by the oxyures.

for Heller[1] had found an oxyuris in the fornices of the vagina, Simons[2] found two specimens in the cervical canal, Vix saw eggs of the organism in the uterus, and Marro[3] found numerous oxyuris eggs in a fibrous cyst situated on the fallopian tube. Hueck's investigations suggest that the oxyuris may even have reached the submucosa; but he has at no time seen sections of the organism in the muscularis, nor found any records of such findings in the literature.

In contradistinction to the above-named authors, Hippius and Lewison write as follows: "At one place the bodies of the oxyuris are only separated from the serosa by means of a thin layer of fiberlike connective tissue." In his recent communication Rheindorf[4] stated that as no inflammatory processes of

[1] Heller. Über Oxyuris vermicularis. Deutsch. Arch. f. klin. Med., 1903, lxxvii, 21.

[2] Simons Entozoen in der Gebärmutter. Zentralbl. f. Gynäk., 1899.

[3] Marro. Cited in the Ref. in Zentralbl. f. Bakteriol. u. Parasitenk., 1902, xxxi.

[4] Rheindorf." Ist die Oxyuris vermicularis im stande aktiv die Processuswand zu durchdringen, und ist sie ein blutsaugender Parasit? Zentralbl. f. Bakteriol. u. Parasitenk., 1914, lxxiv, Part 1.

note were found in the vicinity of the oxyures, it was conceivable that the organisms had in the case mentioned by him burrowed their way actively through the musculature.

It is of course conceivable that the oxyuris is able to penetrate the appendix-wall as far as the serosa by way of the previous pathologically initiated perforation. Whitelocke[1] has recently reported a case in which the abdominal cavity at operation was seen to contain a dozen dead oxyures, together with a quantity of putrefactive and semipurulent fluid; the organisms were covered with omentum. In addition the small pelvis was seen to contain four living oxyures, the processus was inflamed and showed a small perforation, from which during the operation emerged several more worms. We are unable to know whether this was a case of active penetration of the oxyures, for the appendix in question was not examined microscopically. Thus, it is a difficult matter to decide whether perforation in appendicitis is primarily caused by the penetration of the oxyures, or whether the reverse is true; viz., that the oxyures penetrate a perforation already formed.

My own investigations may here be appropriately quoted. In one case, an oxyuris was found in the act of penetrating the mucosa with its head. Under a low-power microscope the section suggests a depression-formation; but under a more powerful lens the mechanical destruction of the tissue at this point can be seen. As already mentioned, depression-formation never results in the destruction of tissue, but in such cases the wall always remains smooth. The site of the perforation was between the two gland-tubes (Fig. 25).

Sometimes the oxyuris lies superficially on the mucosa immediately under the epithelium (Figs. 26, 27, 28, and 29); at other times it lies deep down in the lymph-nodes, extending to the vicinity of the submucosa (Fig. 30). In one case I found a specimen of oxyuris which was boring its way from the lumen into the lymph-nodules (Figs. 31 and 32). This preparation shows a longitudinal section of the anterior part of the oxyuris; the muscularis mucosæ has been completely bored through, and the head end of the organism has reached the lymph-nodules in the vicinity of the submucosa. It is of especial note to observe that the edge of the walls of the mucosa toward the oxyuris is quite smooth and that neither important traumatic destruction, nor inflammatory changes, nor yet hæmorrhage are to be seen in the tissue; all that may possibly be noted is a certain degree of nuclear disintegration in the cells situated at both edges of the duct. The preparation shows a fairly broad space between the worm and the tissue wall, which, however, was produced by the shrinkage of both oxyuris and tissue upon fixation. This leads one to think that before fixation took place there was no space at all, and that the oxyures were consequently in close contact with the tissue.

In my investigations I have never seen cases in which the oxyuris actively bored its passage through the musculature of the intact processus, although this was often found to have occurred in the mucosa and, according to Hueck, in the submucosa as well.

Rheindorf's illustration shows a large muscular defect with a torn wall, filled with fæcal matter; the oxyuris is embedded in the fæces. A small portion of a worm is also seen in the fæces on the peritoneum. Owing to the total absence at the borders of the duct of any inflammatory infiltration and of phlegmonous inflammation of the layers of the wall, Rheindorf feels justified in assuming that this muscular defect was produced by active penetration of the oxyuris. But in my own preparations the wall of the duct is invariably smooth.

This problem may be explained in the following manner: The oxyuris in the case of an absolutely normal appendix usually bores actively through the mucosa, sometimes as far as the submucosa, but in a normal appendix we can only admit of active penetration of the entire musculature when a specimen of the organism is found, together with a smooth wall, as seen in my illustration.

The above-described data taken from my personal observations permit me to affirm with certainty that in post-mortem material the oxyures are found in the lumen, the gland-

[1] Whitelocke. A case simulating meningitis in which the symptoms were caused by the escape of threadworms into the peritoneal cavity through a perforated appendix vermiformis. Brit. J. Child., Dis., 1913, x, 296.

tubes, the mucosa, and at times even in the lymph-nodules, unaccompanied by any noteworthy pathological changes in the appendix, and that in such cases, contrary to the declarations of Rheindorf, who has described various anatomical changes in the post-mortem material, there are no defects, there is no formation of ducts, and no destruction of lymph-nodules, whereas in the extirpated material such changes are always seen.

INVESTIGATIONS IN CONNECTION WITH MATERIAL EXTIRPATED FROM CASES OF APPENDICITIS

Among the 103 extirpated appendices I found 16 cases of oxyuris, 6 of which showed the presence of the organism in the mucosa. It is unnecessary to give a histologic description of the individual cases, for which reason I will confine myself to the following brief mention of certain of the cases which may serve to cast light on the problem at issue.

a. Cases where the presence of the oxyuris in the appendix is to be regarded as purely accidental. The presence of oxyures in the extirpated material is usually to be looked upon as accidental. Although a few oxyures may have been found in the lumen of an inflamed appendix, we are unable to state offhand that such a case of appendicitis has been caused by the parasites. Even in cases where the oxyures are found in the mucosa of an inflamed appendix, it is very hard to decide whether or not this inflammation has been provoked by the worms; for, as has already been shown, these parasites sometimes locate in a perfectly intact appendix. We surgeons even meet cases in which, owing to appendicitis-like symptoms, and the boring pains in the region of the cæcum, a non-inflamed appendix is extirpated, in the lumen of which a few parasites are found. Now, it is almost impossible in the absence of any additional demonstrable pathologic change to decide whether or not this clinically appendicitis-like but non-inflamed condition is caused by the presence in the lumen of several oxyures; for such a symptom might occur equally in an individual whose appendix was free of oxyures.

Case 1 (Fig. 33). In a chronically inflamed appendix, the lumen of which is filled with hard fæcal matter, is seen a transverse section of an oxyuris situated between the mucosa and the fæcal mass. Several large and small fissures seen in the mucosa are to be considered as artefacts, caused by the shrinkage of the tissue during fixation. The entire lumen from the proximal to the distal end is filled with hard fæcal matter. This case can rightly be held to be one of appendicitis chronica stercoralis, and we can at the same time assume that the oxyuris situated between the fæcal mass and the wall of the appendix played no pathological rôle in the causation of this case of appendicitis.

Case 2 (Fig. 34). A chronically inflamed appendix with extensive destruction of the wall, the lumen being filled with liquid fæces. Sections of two oxyures embedded in the fæces are seen near the surface of the mucosa in the vicinity of the defects in the wall. This breakdown of the wall must be regarded as the consequence of chronic inflammation. This defective part was in the first place filled with liquid fæces and the oxyuris doubtless reached this site accidentally. For this reason we must conclude that the oxyures are in nowise connected either with the chronic inflammation or with the destruction of the wall.

Case 3 (Fig. 35). A case of chronic appendicitis, with deep and extensive destruction of the walls which are seen to be perforated. The lumen is filled with hard fæces. We see three oxyures embedded in the mass of detritus and pus, two of which are situated between the fæces and the mucosa in the vicinity of the defects in the wall, and the other at the entrance to the defective part. This case is to be diagnosed as appendicitis chronica purulenta stercolaris, and it is quite evident that the oxyures were in no way concerned in the breakdown of the wall. If they had been instrumental in its destruction they would be found at the same site. This relation is easily seen in Fig. 15, taken from a normal appendix. In the examination of post-mortem material, a worm was sometimes found situated between the fæces and the wall. Now, when such an appendix has become inflamed through any other cause and if in consequence destruction of the wall has occurred, the above result is seen to occur. It is possible of course that a previously existing condition of destruction or some other defect may be aggravated by the oxyures, but I here wish to emphasize that an appendix harboring oxyures was sometimes found showing perforation, not at the point of the location of the oxyures, but at a totally different place.

Case 4 (Fig. 36). Acute suppurative appendicitis with perforation. The lumen is filled with pus. In a purulent infiltrated lymph-nodule are found two oxyuris sections, but it is particularly noteworthy that neither destruction nor perforation has occurred at that site. Several small clefts, which are only artefacts, are seen in the neighborhood of the oxyures. A large perforation is seen on another side of the lumen, where there are no oxyures. In this case, therefore, we must also con-

clude that the presence of the oxyures in the mucosa is purely accidental and that they were not instrumental in the perforation of the appendix.

b. Oxyures as the true causative agent in appendicitis. Is the oxyuris responsible for the causation of appendicitis? Until the present day this question has been actively discussed by the various authors. Of course the direct causative agent of the acute inflammations must be bacterial microörganisms; but we would nevertheless like to discover whether the injuries to the tissues caused by the oxyuris offer a portal of entrance to the bacteria, or whether or not the chronic changes brought about in the tissues by the worm influence the onset of an acute inflammation. To decide this question beyond dispute, it is necessary to have a case which upon thorough histological study will furnish the necessary evidence. In one case characteristic changes were found to have been caused in the wall of the appendix by the oxyuris.

The individual in question was a boy 16 years old showing the symptoms of acute appendicitis. The appendix swelled to the size of a thumb and the serosa was greatly injected. Several oxyures emerged from the proximal end of the lumen. It was also possible to ascertain histologically that the lumen had been stopped up with several oxyures. In the mucosa, sometimes also in the submucosa from the proximal to the distal end, several organisms were found at any point throughout the entire processus, so that the serial sections of the whole appendix showed the presence of the oxyures everywhere (Figs. 37, 38, and 39). The surface of the mucosa was irregularly torn. In the various parts of the mucosa can be seen a necrotic sclerotic change of tissue, several old hæmorrhages in the tissue, and moreover, around the worms an extensive necrotic zone with hæmorrhages. The submucosa, muscularis, and the serosa are infiltrated with polynuclear leucocytes (Figs. 40, 41, and 42).

In one preparation we see only a small transverse section of an oxyuris; but another shows a long vertical section of a worm, which has bored its way from the mucosa to the submucosa (Fig. 43). Some of the oxyures in the mucosa are connected with the lumen. In Fig. 44, we clearly see this relation and this specimen shows a large oxyuris in a longitudinal section placed diagonally to the deeper part of the mucosa, and between this oxyuris section and the lumen a wide passage has been formed in which is situated a small oxyuris cut transversely. The wall of the mucosa about the oxyuris is, for the most part, smooth, and between the worm and the tissue can be seen a fairly wide space, possibly formed by the shrinkage of the tissue upon fixation. From this fact we can imagine that before fixation there was no intervening space at all. One preparation, however, shows an irregularly torn wall containing an oxyuris in section, and in the neighboring tissue recent hæmorrhage and disintegration of cells can be seen. This points to a quite recent traumatic destruction of the tissues through the oxyuris (Fig. 45).

It is conceivable that the oxyuris primarily penetrated the mucosa which resulted in traumatic injury to the tissue around the worm, and thereafter in bacterial inflammation. Of course the possibilities cannot be ruled out that the oxyuris may secondarily penetrate the wall of an appendix which has already undergone inflammatory change, or that in the wall of an appendix which first harbored the worm the inflammation may have been provoked by other causes, unconnected with the presence of the organisms.

According to my experience it is not very difficult to determine histologically whether or not a case of appendicitis is connected with the presence of the oxyuris, for the reason that appendicitis when caused by the worms presents a typically characteristic picture. I will now give a brief description of the important pathologico-anatomical changes brought about in this form of appendicitis. They are as follows:

1. Presence of several oxyures in the wall of the appendix.

2. Necrotic changes accompanied by hæmorrhage in the tissue in the neighborhood of the oxyuris.

3. Extension of the sclerotic degenerated foci in the mucosa.

4. Relatively good preservation of the tissue in the parts of the mucosa uninjured by the oxyuris, which thus presents a great contrast between much disintegrated and the well preserved parts in the same microscopic field.

From this consideration, it can be concluded that the result of the penetration of the oxyuris into the wall of the appendix is to injure the tissue traumatically, and that thence an inflammatory agent enters the tissue and at times may provoke acute inflammation. Such cases of appendicitis caused by the oxyuris show a characteristic histo-

logical picture, for which reason they will be denominated "appendicitis oxyurica." But it should here again be emphasized that this type of the disease occurs but rarely, and in the 103 cases examined by myself but one such example was found, although the organisms are found fairly often in the inflamed appendices.

c. Oxyures as the cause of a non-inflammatory disease of the appendix. In three subjects from whom the appendices were removed on account of the clinical conditions resembling appendicitis, I found that a peculiar change had occurred; viz., extensive destruction of the mucosa without any sign of inflammation.

CASE 1 (Fig. 46). The illustration shows five specimens of oxyuris in the lumen, which contains no fæcal matter, but only a certain amount of mucus. The epithelium of the mucosa is well preserved. There is a pronounced defect in the mucosa, the edge of which is in part covered over with epithelium, and this defect communicates with the lumen by way of a narrow passage. The latter has doubtless been produced by the oxyures. The lymphatic tissue by the side of this large defective place is seen to be filled with a section of an oxyuris. This may lead one to think that the oxyuris had penetrated from the lumen into the lymph-nodules through the narrow passage and the large defects; but I do not think that the entire space of these defects was formed by the worm, as the wall of the same was partially covered over by epithelium. Upon examination of the appendix one sometimes finds a diverticulum-like physiological depression of the mucosa. This fact enables us to understand the nature of this space. The oxyuris has penetrated through the narrow passage into the depression already present and, after destroying a part of the same, has entered the lymph-nodule.

In the following series of sections (Fig. 47) the passage is no more visible; but here the oxyuris section in the lymph-nodule is seen much more distinctly than in the previous illustration, and on one side of the large space is seen a row of well-preserved epithelium, while in the remaining part of the same no epithelium is visible. Rheindorf hinted that such a formation of the mucosa in the fissure was a case of regeneration, but I believe that if such were the case the scar formation would be seen.

In addition to these traumatic disturbances of the mucosa a certain amount of old hæmorrhage is visible in the neighborhood of the oxyuris, but apart from this there is no noteworthy change and no inflammation. Thus, it is conceivable that in this case the passage and the defects were much smaller before fixation than they appear in the preparation.

CASE 2 (Figs. 48 to 52). In the first section of the series, we find two worms in the lumen of the appendix surrounded by blood; no fæcal matter is present and the epithelium is well preserved. The next section shows an oxyuris in the act of penetrating the mucosa, and in the remaining series the worms in the lymph-nodules are seen almost to have reached the submucosa. It is especially noteworthy that the blood is found not only in the lumen, but also in the tissue of the submucosa in the neighborhood of the oxyures. In the entire series of sections no trace of inflammation is seen.

It is of course possible that such a bleeding occurs traumatically during operation, but I believe that it is provoked by the oxyures, as this site corresponds to the middle of the appendix and no blood can be seen in the remaining part of the processus. It is inconceivable that during operation a circumscribed focus of hæmorrhage can be produced, confined to that part in which the oxyuris is harbored, and that it can be produced nowhere else.

CASE 3 (Figs. 53 to 64). In this case we clearly see the extensive destruction of the mucosa brought about by the oxyures. The lumen contains neither oxyuris nor fæces, but only mucus is seen. The epithelium is well preserved. In the first series can be seen a large fissure with hæmorrhage in the lymphatic tissue of the mucosa, beside which is a small oxyuris section. The edge of this fissure is in part sharply outlined and in part irregularly torn. In the other series the oxyures are found in the lymph-nodules which were greatly injured by the worms. At the site of the oxyures a fissure has been formed, accompanied by hæmorrhage; the wall of the fissure in the lymph-nodule is for the most part torn. The last of the series shows a large defect in the lymph-nodule where there is no remaining part of the oxyuris, but this is an oxyuris fissure. If this series be compared with the preceding section we are at once able to understand that it is a lymph-nodule which has been destroyed by the oxyures. Close to this large fissure there are some more smaller clefts with hæmorrhage, which must be considered as cases of tissue destruction produced by the organisms, as they are accompanied by bleeding, which does not occur in fissures due to artefacts.

From the consideration of this specimen I was able to ascertain that pseudo-appendicitis could be provoked by the oxyures even without the incidence of inflammation. Hueck already designated a painful pathological condition of the appendix, other than appendicitis, by the name "appendicopathia oxyurica," but among the 18 cases examined by him he found the parasite but once in the

mucosa and submucosa; it was usually found only in the lumen. It is doubtful whether the organisms when situated in the lumen are indeed able to produce a morbid condition similar to appendicitis without the accompaniment of any demonstrable anatomical changes. My own experience leads me to believe that an individual can, with impunity, harbor the organisms in the appendix, without the necessary accompaniment of any abnormal symptoms. It was especially important to note that in post-mortem material the oxyures were situated in the wall of the processus, without any demonstrable histological changes having been effected; whereas in the cases of extirpated appendices extensive traumatic destruction was always present. In women, especially, we often find morbid appendicitis-like conditions, unaccompanied by infection or by the presence of oxyures.

Of course it is absolutely impossible to discover whether a given pain has been caused by the presence of oxyures in the lumen.

Since I could always demonstrate in my cases a traumatic destruction of the mucosa accompanied by hæmorrhage, I will designate such a case by the name of "appendicopathia oxyurica traumatica."

What is the reason, then, that in the surgically extirpated appendices we invariably find traumatic changes, while in the post-mortem material no reaction is seen around the oxyuris? It appears that this circumstance is connected with the life-energy of the parasite. If the worm remains inserted in the gland-tube or if, after having penetrated to the mucosa, it ceases all violent movement, no demonstrable changes occur and in such cases all clinical symptoms remain absent.

SUMMARY

1. In an appendix the oxyures may be harbored, not only in the lumen, but also in the mucosa and submucosa; this may occur without producing any clinical symptoms or any noteworthy anatomical changes.

2. We sometimes encounter a case in which an appendix infected with oxyures is inflamed through a totally different agent. For this reason the origin of appendicitis cannot be attributed to the worms, although these parasites have been found in an inflamed appendix. In the majority of cases the presence of the oxyures in cases of appendicitis, whether it be chronic or acute, is to be considered as a pure accident.

Rheindorf always attributed too great importance to infection caused by oxyures. His statement to the effect that all cases of appendicitis or pseudo-appendicitis are to be attributed to oxyuris infection, appears to me to be inaccurate.

3. I was able to ascertain that a true inflammation is provoked by the parasites when the latter have penetrated the wall of the appendix in large numbers, and that the traumatically destroyed fissure in the tissue is for a protracted period of time in connection with the lumen, thus offering the infecting agent a portal of entry. This type of the disease offers us distinct histological data, for which reason I have designated it "appendicitis oxyurica." But this type of the disease is only rarely met, and among 103 surgically extirpated appendices I met but one such case.

4. A non-inflammatory, but pain-producing morbid condition of the appendix is sometimes caused by the oxyuris, in which cases traumatic destruction of the tissue accompanied by hæmorrhage can always be demonstrated. It is very much to be doubted whether oxyures which are merely situated in the lumen are able to cause this painful condition, without any accompanying anatomical changes.

Among the 103 above-quoted cases I found three cases of this type.

5. From my investigations I am able to affirm with certainty, that a part of the oxyuris passage and cleft of which Rheindorf speaks, and several fine fissures in the lymph-nodules seen in his illustrations, as well as certain defects at the edge of the mucosa, where no oxyures are to be found, are to be looked upon as artefacts. But on the other hand, Aschoff is, in my opinion, mistaken in considering every oxyuris passage and fissure to be purely artificial in origin, since the evidence of my own preparations, presented in this paper, must lead one to admit that an undoubted cleft is occasionally formed by the parasite.

METHODS EMPLOYED FOR DETERMINING THE HYDROGEN-ION CONCENTRATION IN BODY FLUIDS

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From the Clinic of Dr. George W. Crile, Lakeside Hospital, Cleveland

PRESENT interest in the problems of acid production and control within the body, and in the known clinical concomitants of acid intoxication, constitutes a sufficient excuse for these notes on devices employed for the measurement of the hydrogen-ion concentration (true acidity) in physiologically important fluids. This is especially true since many recent improvements in technique have not been made available in collected or easily accessible form. Some understanding of purely physicochemical matters is essential for the proper appreciation of these methods, and, indeed, for the interpretation of the results as well. Progress has recently been made, however, in the development of methods which are at once rapid, easily carried out, and sufficiently accurate for certain kinds of routine work.

The results which have been secured in this field are of considerable interest to the physiologist, but in some respects their possible practical significance has yet to be established.

It is not possible to give in this place any summary of the published hydrogen-ion concentration measurements relating to body fluids. The recent monograph of Michaelis (1) is very complete up to the time of its publication, and references will subsequently be made to other and more recent articles.

These notes refer mainly to the technical procedures which we are using in the laboratory of Dr. G. W. Crile for the study of problems concerning "acidosis."[1] Except in regard to some details, nothing original is claimed for these methods; but because they have proved most useful in several months' continuous work necessitating a large number of hydrogen-ion determinations, it is hoped that they may be of service to others.

A word is perhaps required regarding the notation used in recording hydrogen-ion concentrations. It is customary to express these values in a convenient way suggested by Sörensen, namely by the *logarithm* of the hydrogen-ion normal (C_H), with the negative sign omitted; this is denoted by the symbol P_H. Inasmuch as we are dealing with ionic concentrations which are far below normal, this exponent P_H is negative, and an increase in P_H denotes a decrease in the hydrogen-ion content. This may be illustrated by the figures in Table 1, which also serves to show the comparatively large significance of small variations in P_H.

TABLE I

$(H^+) \times 10^{-7}N$	P_H
0.1995	7.70
0.2512	7.60
0.3162	7.50
0.4000	7.40
0.6300	7.20

It is, also, not out of place to call attention briefly to the possible significance of the hydrogen-ion content of body fluids and the variations in this value. Evidence has been found, and is rapidly being increased, which shows that the activity of the respiratory center is controlled by the P_H of the blood, in its turn normally determined by the concentration of carbonic acid. It is further quite possible [2] that the hydrogen-ion unites directly with the hæmoglobin molecule, thus interfering with oxygen transportation, in addition to affecting the physical condition of hæmoglobin in other ways. More important than this, in some respects, is the known dependence of the activity of enzymes upon the P_H of their media; since there has recently been opened up the complex possibility that the action of enzymes, and conceivably of

[1] "Acidosis" is a much abused word; indeed, one incapable of exact definition, in view of the diverse and frequently very vague usages to which it has been subjected. Probably the best working idea of "acidosis" may be obtained from the paper of Palmer and Henderson (2), wherein it is proposed to consider that a condition of acidosis exists when the bicarbonate depletion of the blood has been carried to such a point that the administration of a quantity of sodium bicarbonate equivalent to 1 liter of n/10 solution fails to diminish the urinary acidity.

[2] cf. Barcroft, J., 1914. The Respiratory Function of the Blood (Cambridge), p. 316.

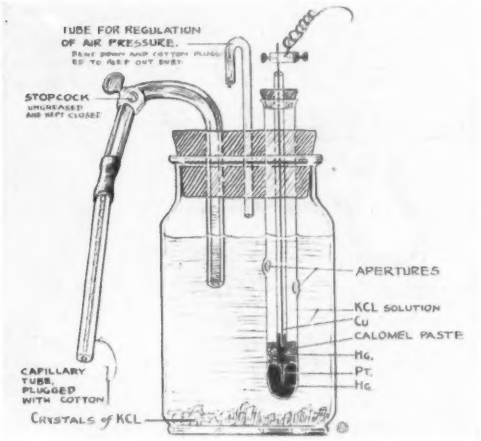

Fig. 1. Barendrecht's electrode.

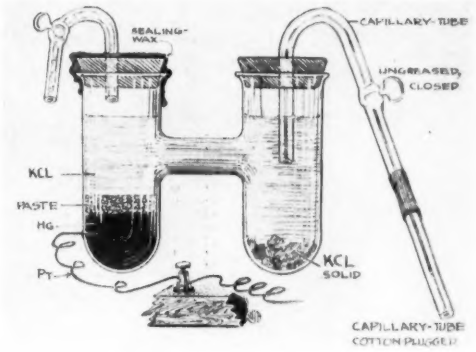

Fig. 2. New form of electrode.

hormones also, is the result of a balance between the "active agent" itself and various inhibitors, it should be taken into account that irregularities in P_H may be responsible for various abnormal manifestations of enzyme or hormone effects Linked with this matter is the further probability that hydrogen-ion variations result in changes of the aggregation condition of the colloidal [1] materials of cell membranes thus being implicated in important modifications in the permeability of cells.

I. *The gas electrode.* The fundamental method of acidity determination is based upon the fact that the potential developed at the contact of gaseous hydrogen with a solution containing the hydrogen-ion is dependent, among other things, upon the concentration of the hydrogen-ion within that solution. Under controlled conditions it is possible to calculate the hydrogen-ion concentration from the measured potential of a "gas electrode" of which the essential constituent is a layer of colloidal platinum black, usually deposited on a P wire (or on glass), which is saturated with hydrogen [2] and part of which is immersed in the solution being studied. This electrode forms one "leg" of a battery, the other limb being most usually a calomel electrode containing KCl as electrolyte. The potential of this battery is then measured by the compensation method, using resistance boxes and a capillary electrometer of the enclosed (Luther) type. As the source of compensating electromotive force a lead accumulator or a couple of dry cells may be employed. The value of this opposing electromotive force is found by comparison with a Weston normal cell.[3]

The two points requiring most delicate consideration are the calomel electrode and the hydrogen element. Many types of calomel electrode have been suggested, but we are finding most serviceable two forms which differ slightly from the ones pictured in many textbooks. The first of these is that designed by Barendrecht (4), and the second is a form of electrode, which has proved even more generally satisfactory (see Figs. 1 and 2), constructed with the H-vessel familiar in standard cells. These electrodes have an especial advantage for electrometric titrations. It is best for routine work to use the saturated KCl-calomel electrode; a tabular statement of its characteristics will be found in Michaelis' book (1). It is very convenient to have two calomel electrodes which may be used alternately in duplicate determinations. The calomel electrodes may be standardized either by comparison with a "null" electrode,

[1] It is known that variations in P_H accompany the coagulation of proteins and other colloidal solutions (cf. Walpole, 3).

[2] This combination acting like a conducting form of H_2.

[3] For the technical details of these procedures, see Findlay's Practical Physical Chemistry; or Michaelis (1).

All connecting wires should either be shielded with glass tubing or have the insulations grounded. This is to avoid the very insidious effects of stray currents.

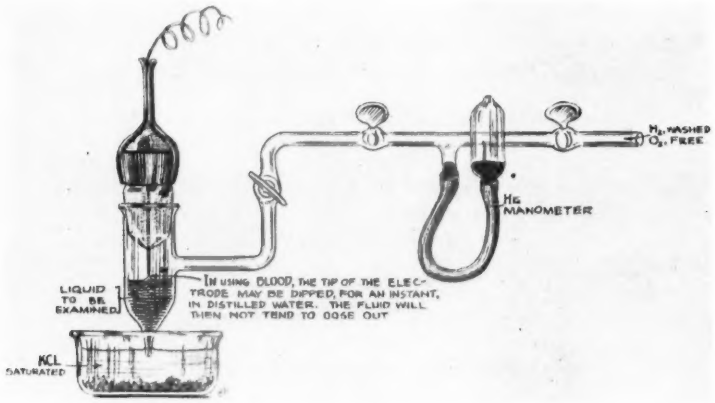
H₂ WASHED O₂ FREE
Hg MANOMETER
LIQUID TO BE EXAMINED
IN USING BLOOD, THE TIP OF THE ELECTRODE MAY BE DIPPED, FOR AN INSTANT, IN DISTILLED WATER. THE FLUID WILL THEN NOT TEND TO OOZE OUT
KCL SATURATED

Fig. 3. Hydrogen electrode, modified from Walpole.

or, more conveniently, against solutions of known P_H. It is possible to construct also curves relating the resistance in the hydrogen battery circuit when it is exactly balanced to the P_H at a given temperature. These curves are useful in a double way; namely, in standardizing the electrode and in affording a very decided short cut in the calculation of P_H in "unknowns."

With physiological fluids the loosely contained CO_2 is a very considerable source of error in hydrogen-ion determinations, since it may so easily be lost by exposure to the air or by the bubbling through of hydrogen or other gases. Several more or less laborious schemes have been devised to overcome a possible increase in alkalinity (of blood, for example), by the loss of CO_2. By far the most practical of these methods is that suggested by Michaelis, namely the use of a *small* stationary volume of hydrogen with minimum contact between the platinized electrode and the solution which is being analyzed.[1]

The small contact surface between platinum and solution assures that only a small volume of blood must be reduced. The reduction is essential if one is to avoid oxidation potentials which interfere decidedly with the readings of electromotive force for the gas cell. Michaelis' electrode is very useful for work on blood, but the Walpole (5) electrode is very much less cumbersome and more generally handy for determinations on many kinds of fluids. It is believed better to substitute for the plunger, which Walpole uses for filling, a manometer attachment. (Fig. 3)[2]

The connection between the gas and calomel electrodes is best made through a saturated KCl bath,[3] or, in many instances, the cotton filled tube of the KCl-calomel electrode may dip directly into some of the fluid which is being analyzed.

Having measured the potential of the hydrogen battery, the hydrogen-ion content is calculated from Nernet's formula:—

$$\pi_p = \pi_o + 0.0577 \log. \frac{Co}{Cp}$$

π_p = Electromotive force of "unknown" fluid.
π_o = Electromotive force of the normal hydrogen electrode.

The value of π is substituted corresponding to the temperature at which the measurements are being carried out, and C_p may then be calculated: P_H may be obtained from the expression

$$P_H = \frac{\pi_p - 0.3377}{0.0577} \text{ (at 18° c).}$$

II. *Indicators.* The use of indicators for hydrogen-ion estimations is open to at least three sources of error; namely, the influence of neutral salts, or amphoteric proteins, and of the color and opacity or turbidity of the fluids examined. Without going into many

[1] See Walpole (6).

[2] Barendrecht's gas cell has not, in our hands, proved satisfactory.

[3] For theoretical reasons which cannot be treated of in this place.

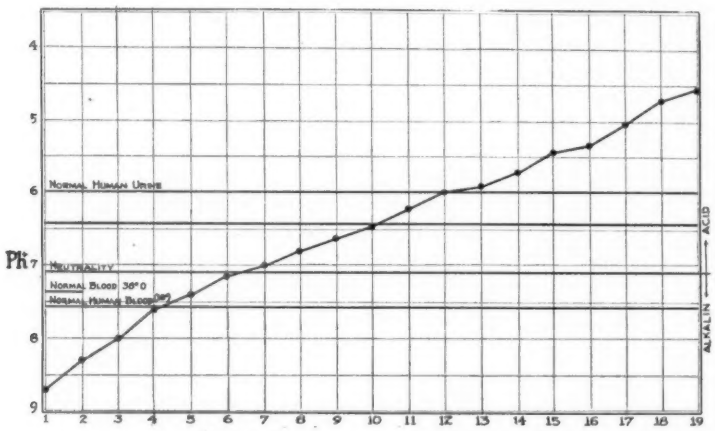

Fig. 4. Chart for [H] estimation — 18°. The solution number is shown at bottom of chart.

details, it is sufficient to say that, with a properly prepared set of standards for comparison, it is possible to utilize indicators for the estimation of the P_H of urine, blood, cerebrospinal fluid, bile, milk, and various other aqueous solutions. The wide application of the indicator method is made possible by dialysing against an appropriate fluid such things as blood, milk, and the like, to which the indicators may not be directly applied.

A. *Direct use of indicators.* This method has been successfully applied by Henderson (2) and others to the study of acid excretion in urine. We find (see subsequent tables) that it may also be used for the determination of P_H in cerebrospinal fluid.

The indicators we employ are two:

1. Sodium alizarin sulphonate (2 per cent solution).

2. Phenolsulphonephthalein (0.6 per cent solution).

These indicators are used in combination, one drop of each in the tubes of the standard set and in the "unknown"; test tubes of uniform caliber are used as containers. The solutions of known P_H are given in Table II; 5 ccm. of each of these is diluted in a test tube to about 15 ccm. after the addition of the indicators. The indicators are added from pipettes used throughout for this purpose. The indicators may be kept mixed and the drop added from a stalagmometer. It is essential, or at least very desirable, that these standards be checked by gas chain determinations. The set of comparison tubes is stable for some days. The standard stock solutions are stored in hard glass flasks. Two cubic centimeters are sufficient for an analysis, the dilution reducing to a minimum the error from neutral salts and the color of urine.

TABLE II

Solutions:—

Designation	Component	Concentration.
A	Sodium acetate ($NaC_2H_3O_2=3H_2O$)	N/5 (27.20 gms. liter)
B	Acetic acid	N/5
C	Disodium phosphate ($Na_2HPO_4=12H_2O$)	N/10 (17.8 gms. liter)
D	Monopotassium phosphate (KH_2PO_4)	N/10 (13.6 gms. liter)

Solutions: (Proportions make up to 50 cc.+, dilute to 250)

					P_H
1.	0.5	ccm. (d) +	240	ccm. (c)	8.7
2.	9.7	ccm. (c) +	0.3	ccm. (d)	8.3
3.	0.5	ccm. (d) +	60	ccm. (c)	8.00
4.	9	ccm. (c) +	1	ccm. (d)	7.60
5.	5	ccm. (d) +	25	ccm. (c)	7.40
6.	7	ccm. (c) +	3	ccm. (d)	7.15
7.	5	ccm. (d) +	11.5	ccm. (c)	7.00
8.	5	ccm. (c) +	5	ccm. (d)	6.80
9.	4	ccm. (c) +	6	ccm. (d)	6.63
10.	3	ccm. (c) +	7	ccm. (d)	6.40
11.	2	ccm. (c) +	8	ccm. (d)	6.23
12.	1.15	ccm. (c) +	8.85	ccm. (d)	6.00
13.	1	ccm. (c) +	9	ccm. (d)	5.92
14.	1	ccm. (b) +	10	ccm. (a)	5.74
15.	1	ccm. (b) +	7	ccm. (a)	5.45
16.	1	ccm. (b) +	4	ccm. (a)	5.35
17.	1	ccm. (b) +	2	ccm. (a)	5.05
18.	1	ccm. (b) +	1	ccm. (a)	4.74
19.	1.5	ccm. (b) +	1	ccm. (a)	4.57

(Av = 0.25 per step)

A graph illustrating the P_H values of the standard solutions is appended (Fig. 4). The curve serves a special purpose, in that it is found possible, after some practice, to interpolate with a very fair degree of accuracy when the "unknowns" do not exactly coincide with any of the comparison tubes.

It is worth while to call attention to the fact that this indicator series in combination with the use of *two* indicators affords a scale of colors which shows good differentiation between the several steps in the series over a wide range of P_H values.

B. *Dialysis.* Notes on dialysis as applied to hydrogen-ion concentration estimation in blood, bile, turbid urine, etc.

Dialysis membrane — collodion sacs. A satisfactory membrane for the procedure has been prepared as follows: A 10 per cent collodion [1] (E. Schering) is made by dissolving 10 gms. of the collodion in ether 50 ccm. and ethyl alcohol 50 ccm. complete solution being obtained in 48 hours. Allow the preparation to stand, agitating at intervals.

About 2 to 3 ccm. of the collodion solution is poured into a good sized test tube, the tube held horizontally and rotated slowly until the entire inside of the tube is coated. The test tube is then inverted and any excess of collodion allowed to run out. The tube is held inverted in the hand until the ether is driven off and then reversed and filled with warm water. This step seems to be one of the most important in the technique, because if left to dry too long the membrane is hard, brittle, and more impervious. If immersed too soon the sac is thick, opaque and not tough, and may even contain bubbles. The point of time at which it is best to immerse the preparation may conveniently be determined by the odor from the tube. Just when ether can no longer be detected and alcohol begins to come off, the tube should be filled with warm water and allowed to stand for a minute. The sac may then be very gently separated from the inside of the glass. A very thin, transparent, rather tough, permeable membrane will be obtained. The sacs are kept immersed in a jar of distilled water until used.

[1] It is important that the collodion be neutral, or as neutral as possible.

Five to ten cubic centimeters of the solution to be dialysed is placed in one of these membranes, which is then lowered into a short tube of large diameter, containing about 15 cubic centimeters of normal saline solution at body temperature. The whole is then kept at 37° C. for about five minutes, the dialysate poured off, and the determination made after cooling to room temperature. The readings made by two separate workers have been found to correspond very accurately with those made with the gas chain.

As found by Rowntree (7) either whole blood or serum may be used, readings being the same. For the dialysis of milk, turbid urine, etc., distilled water may be used in the outer tube.

III. *The oxyhæmoglobin dissociation curve as an index of acidosis* (Barcroft method). An indirect method of estimating the alkalinity of the blood, or rather of detecting the existence of acidosis, has recently been employed by Barcroft and his co-workers. This is based upon the fact that the affinity of reduced hæmoglobin for oxygen is dependent upon the reaction of the blood, being decreased with increasing acidity, probably as the result of direct interference on the part of the hydrogen-ion with the oxygen fixing power of hæmoglobin. A condition of acidosis exists when the blood has less than its usual (normal) affinity for oxygen (meionexy). The degree of meionexy is expressed by the value of the mass action constant (K) in Hill's equation for the dissociation curve of oxyhæmoglobin. This figure K varies inversely with the acidity. In practice this method is attended by several difficulties, which interfere with its routine use in the hands of people unskilled in gas analysis. A small sample (1 to 2 ccm.) of the blood to be examined is shaken at body temperature in a closed vessel (tonometer) containing known partial pressures of nitrogen, oxygen, and carbon dioxide, the latter being present at a tension equal to that found on analysis of the alveolar air. The blood sample is then analyzed for O_2 saturation by the differential method (see Lewis, Ryffel, Wolf, Cotton, and Barcroft, 8).

The method has been applied to the esti-

mation of the acidosis due to non-volatile acid on the basis of comparisons with O_2 acidity measurements upon CO_2 free blood to which various quantities of lactic acid had been added. In this case the technique is the same as in the preceding except that the O_2 saturation is estimated in the presence of a known (constant) tension of O_2 in the absence of CO_2. The non-volatile acid is calculated as lactic acid.

This procedure yields figures which show more variation in the value of K than has been obtained in direct gas chain measurements of P_H. The two types of analysis are, on practical grounds, somewhat difficult to compare, for reasons which may not be gone into here. For example, Lewis, and Barcroft record values of K in cardiac dyspnœa which, on the basis of Peters' measurements of the relation of P_H to CO_2 tension in normal blood (Barcroft), may be calculated to correspond to P_H (at 37°) = 8.93, 7.14, 7.32, etc.

Now a large number of carefully executed determinations have been carried out upon the reaction of the blood in a variety of diseases, and aside from a variation which is readily accounted for on the basis of individual peculiarity there are only a very few instances in which P_H varies by a notable amount from the mean 7.32 (38°) given by Michaelis and none in which (for conditions similar to those studied by Barcroft and Lewis) the variation is so great as that calculated above.

There seems very little doubt that this method affords a valuable index of acid intoxication, whether or not it gives the true hydrogen-ion concentration in blood, but the nature of the technique prohibits its very general use.

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END-RESULTS IN 242 CASES OF SIMPLE FRACTURE OF THE FEMORAL SHAFT[1]

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From the Mayo Clinic

In most hospital statistics, and too frequently in those of private practice, histories of simple fracture of the femoral shaft include only the cause, nature, and symptoms of injury, deformity at the time of admission, method of treatment, and date of the patient's discharge with firm bony union. At this time the word "cured" is simply noted on their discharge blank with no mention in most cases of the amount of deformity or the limitation of motion. In reality the forty-ninth or fifty-sixth day when the adult leaves the hospital on crutches, and in the majority of instances utterly crippled so far as the regular pursuit of his vocation is concerned, signifies the time at which his troubles begin.

The records of these cases are not to be found in a study of surgical literature. We have become so habituated to the word cure in regard to fractures as denoting union, and have so completely lost sight of the need for attaining complete function that we have unconsciously accepted the doctrine that all simple fractures of the shaft of the femur are cured in six to eight weeks. In many of the

[1]Studies completed in 1914. Presented for publication May 18, 1915.

CHART I. END-RESULTS IN SIMPLE FRACTURE OF FEMORAL SHAFT

No.	Name	Age	Sex	Date of Fracture	Location	Primary Shortening	Treatment	Duration of Extension	Bed	Chair	Crutch	Cane	Condition at Discharge	Present Condition
1	T. F.	1 yr.	M	6-25-10	Upper and middle ⅓	0	Buck's extension overhead	6 wks.	6 wks.	0	0	0	No shortening, no limitation of motion	Complete recovery.
2	R. C.	2 wks.	F	7-3-11	Center of femoral shaft	0	Plaster cast	4 wks.	4 wks.	0	0	0	No shortening, no limitation of motion	Complete recovery.
3	M. J.	9. mos.	F	2-9-07	Junction of upper and middle ⅓	0	Buck's extension overhead	5 wks.	5 wks.	0	0	0	No shortening, no limitation of motion	Complete recovery.
4	F. F.	7 mos.	M	5-3-13	Middle of femur	0	Buck's extension overhead	4 days	4 days	0	0	0	Cast for 4 weeks	Complete recovery.
5	F. K.	2 yrs.	M	5-9-12	Lower end, upper ⅓	0	Buck's extension overhead	3 wks.	3 wks.	Cast 2 wks.	0	0	Cast on	Complete recovery.
6	C. G.	1.5 yrs.	F	2-17-10	Lower ⅓ right femur	0	Plaster cast	4 wks.	4 wks.	0	0	0	Cast on	Complete recovery.
7	F. T. G.	14 mos.	M	12-25-09	Lower 1.3 right femur	0	Buck's extension overhead	3 wks.	3 wks.	0	0	0	Good union, no shortening	Complete recovery.
8	A. S.	2 yrs.	M	9-24-13	Upper ⅓ right femur	0	Buck's extension	7 wks.	7 wks.	0	0	0	No shortening, no limitation of motion	Complete recovery.
9	M. K.	2 yrs.	F	8-15-06	Supracondylar	0	Buck's extension from above	5 wks.	5 wks.	0	0	0	Complete recovery	Complete recovery.
10	E. H.	1.5 yrs.	F	3-1-08	Middle of shaft	0	Buck's extension and cast. Open operation	5 mos.	5 mos.	2 days	0	0	No shortening, no limitation of motion	Complete recovery.
11	G. H.	1.5 yrs.	F	4-13-08	Middle ⅓ right femur	0	Buck's extension	5 wks.	5 wks.	1 day	0	0	No shortening, no limitation of motion	Complete recovery.
12	A. F.	4 mos.	M	9-9-07	Juncture of upper and middle ⅓ femoral shaft	0	Plaster cast	0	0	0	0	0	Cast on	Complete recovery.
13	R. J.	2 yrs.	F	9-14-10	Simple green stick	0	Plaster cast	6 wks.	9 wks.	0	Ever since	0	Same length	1½ in. short. Right leg paralyzed. Crutches.
14	H. D.	2 yrs.	F	12-2-07	Upper and middle ⅓ right femur	0	Buck's extension	18 days	18 days	0	0	0	Good, firm union, considerable angulation	Braces, no shortening, pain, or tenderness.
15	P. G.	7 yrs.	M	5-14-13	Lower ⅓ right femur	0	Buck's extension	6 wks.	6 wks.	1 day	1 wk.	2 days	Crutches	Complete recovery.
16	J. F.	3 yrs.	M	7-6-12	Middle of shaft of right femur	0	Buck's extension	6 wks.	6 wks.	0	0	0	Weakness, no shortening	Complete recovery.
17	F.McD.	5 yrs.	M	5-21-06	Upper ⅓ left femur	0	Buck's extension	7 wks.	7 wks.	1 day	0	0	Weakness, no shortening, no limitation of motion	Complete recovery.

18	G. J.	6.5 yrs.	M	10-5-11	Supracondylar	0	Buck's extension	7 wks.	7 wks.	1 day	0	0	Weakness, no shortening	Complete recovery.
19	E. H.	7 yrs.	M	11-5-09	Lower ⅓	0	Buck's extension	6 wks.	6 wks.	0	0	0	Weakness, no shortening	Complete recovery.
20	J. R.	7 yrs.	M	7-5-10	Middle right femur	0	Buck's extension	8 wks.	8 wks.	0	0	0	Weakness, no shortening, no stiffness	Complete recovery.
21	I. T.	7 yrs.	M	1-1-07	Lower end upper ⅓	0	Buck's extension	7 wks.	7 wks.	0	0	0	No shortening, no limitation of motion	Complete recovery.
22	B. B.	11 yrs.	F	11-21-11	Middle right femur	0	Buck's extension	6 wks.	6 wks.	1 day	2 wks.	1 wk.	Crutches. Weakness, no shortening	Complete recovery.
23	M. G.	6 yrs.	M	10-26-07	Upper end lower ⅓ right femur	0	Buck's extension	7 wks.	7 wks.	0	0	0	Good, no shortening	Complete recovery.
24	W. W.	4 yrs.	M	9-13-11	Lower end upper ⅓ right femur	0	Buck's extension	6 wks.	6 wks.	1 day	0	0	No shortening, no limitation of motion	Complete recovery.
25	A. L.	5 yrs.	F	2-15-13	Lower ⅓	0	Buck's extension	11 wks.	11 wks.	1 day	0	0	Weakness, no shortness, no limitation of motion	Complete recovery.
26	A. K.	10 yrs.	M	5-15-13	Lower ⅓ femur	0	Buck's extension	3 wks.	3 wks.	0	2 wks.	0	Cast, no shortening	Complete recovery.
27	P.McD.	10 yrs.	M	7-16-09	Middle of shaft	0	Buck's extension	7 wks.	7 wks.	1 day	0	2 wks.	Cane, no shortening, no limitation of motion	Complete recovery.
28	P. L.	9 yrs.	M	3-24-09	Upper end, lower ⅓ femur	0	Buck's extension	5 wks.	5 wks.	1 day	0	2 wks.	No shortening, no limitation of motion	Complete recovery.
29	M. S.	7 yrs.	F	12-23-06	Lower end, upper ⅓ femur	0	Buck's extension	7 wks.	7 wks.	2 days	1 mo.	0	Crutches and cast	Complete recovery.
30	F. G.	8 yrs.	M	6-26-11	Middle, left femur	0	Buck's extension	6 wks.	6 wks.	0	0	0	No shortening, no limitation of motion	Complete recovery.
31	S. R.	6 yrs.	M	11-21-06	Shaft, right femur	0	Buck's extension	7 wks.	7 wks.	0	0	0	Some weakness, no shortening	Complete recovery.
32	A. B.	4 yrs.	M	6-25-13	Transverse fracture middle shaft, right femur	½ in.	Cast for 3 wks., then Buck's extension	4 wks.	7 wks.	0	0	0	Walked well	No shortening. Complete recovery.
33	A. N.	9 yrs.	M	5-29-05	Transverse fracture middle and upper ⅓ left femur	½ in.	Buck's extension	8 wks.	8 wks.	2 days	3 days	0	Crutches, ½ in. shortening	Complete recovery.
34	H. D.	7 yrs.	M	2-2-06	Juncture middle and lower ⅓	0	Buck's extension	8 wks.	8 wks.	0	0	0	O. K., No shortening	Complete recovery.
35	F. H.	11 yrs.	M	12-19-13	Middle ⅓ right femur	¼ in.	Plaster cast	0	1 wk.	3 wks.	0	2 wks.	No shortening, weakness, no limitation of motion	Complete recovery.
36	A. F.	5 yrs.	M	9-30-07	Middle ⅓ femur	0	Buck's extension	4 wks.	4 wks.	0	0	0	No shortening, walked with limp	Complete recovery.
37	W. R.	4 yrs.	M	7-1-10	Middle ⅓ femur	0	Buck's extension	5 wks.	5 wks.	0	0	0	No shortening, walked with limp	Complete recovery.
38	T. B.	10 yrs.	M	12-17-10	Upper ⅓ of shaft of left femur	0	Buck's extension	10 wks.	10 wks.	1 day	1 wk.	0	Crutch, no shortening	Complete recovery.
39	J. E.	9 yrs.	M	6-23-09	Middle of shaft of right femur	0	Buck's extension	5 wks.	5 wks.	0	0	0	Walked out. No shortening, no limitation of motion	Complete recovery.

CHART I. END-RESULTS IN SIMPLE FRACTURE OF FEMORAL SHAFT—*Continued*

No.	Name	Age	Sex	Date of Fracture	Location	Primary Shortening	Treatment	Duration of Extension	Bed	Chair	Crutch	Cane	Condition at Discharge	Present Condition
40	F. T.	8 yrs.	M	12-6-13	Middle of shaft of left femur	0	Buck's extension	6 wks.	6 wks.	0	0	0	No shortening, weakness, some limp	Complete recovery.
41	T. D.	12 yrs.	M	2-26-09	Upper ⅓ right femur	0	Buck's extension	7 wks.	7 wks.	0	2 wks.	0	Crutches, no shortening, no limitation of motion	Complete recovery.
42	A. N.	8 yrs.	F	3-2-10	Middle of shaft of right femur	0	Buck's extension	10 wks.	10 wks.	1 day	0	0	Weakness, no shortening, no stiff knee	Complete recovery.
43	N. S.	4 yrs.	M	6-25-12	Lower ⅓	0	Buck's extension	7 wks.	7 wks.	0	0	0	No shortening, no limitation of motion	Complete recovery.
44	J. H.	9 yrs.	M	5-2-07	Lower ⅓ of shaft of right femur	0	Buck's extension	8 wks.	8 wks.	0	0	0	No shortening, no limitation of motion	Complete recovery.
45	J. C.	5 yrs.	M	11-3-07	Middle of shaft	0	Buck's extension	8 wks.	8 wks.	1 day	0	0	No shortening, no limitation of motion	Complete recovery.
46	J. C.	6 yrs.	M	9-26-11	Left femur, supracondylar	0	Buck's extension	8 wks.	8 wks.	0	0	0	Limp, no shortening	Complete recovery.
47	H. M.	6 yrs.	M	9- 05	Middle of femur	0	Buck's extension	8 wks.	8 wks.	1 day	0	0	Weakness, no shortening	Complete recovery.
48	P. P.	8 yrs.	M	3-25-12	Middle shaft	0	Buck's extension	6 wks.	6 wks.	1 day	0	0	No shortening, no limitation of motion	Complete recovery.
49	C. A.	4 yrs.	M	12-20-13	Lower ⅓ shaft right femur	0	Buck's extension	19 wks.	19 wks.	2 wks.	0	0	No shortening, no limitation of motion	Complete recovery.
50	J. M.	12 yrs.	M	9-12-10	Juncture upper and middle ⅓ right femur	0	Buck's extension	10 wks.	10 wks.	2 days	4 wks.	0	Crutches, no shortening	Complete recovery.
51	W. C.	9 yrs.	M	4-20-13	Upper ⅓ of shaft of right femur	½ in.	Buck's extension	6 wks.	6 wks.	0	1 mo.	0	Crutches, ½ in. shortening	Complete recovery.
52	J. S.	9 yrs.	M	5-25-07	Middle shaft of right femur	0	Buck's extension	8 wks.	8 wks.	2 days	3 mos.	0	Stiffness, no shortening	Complete recovery.
53	J. C.	3 yrs.	M	11-27-07	Middle and lower ⅓ right femur	0	Buck's extension	9 wks.	9 wks.	4 days	0	0	Could not walk well, no shortening	Complete recovery.
54	L. N.	7 yrs.	M	8-26-12	Upper ⅓ of femur	0	Buck's extension	7 wks.	7 wks.	0	0	0	Cast, weakness, no shortening	Complete recovery.
55	H.McG.	12 yrs.	M	8-2-11	Shaft of femur, subtrochanteric	0	Buck's extension	6 wks.	6 wks.	1 day	2 wks.	0	Crutches, no shortening	Complete recovery.
56	E. M.	5 yrs.	M	9-28-10	Middle of shaft of left femur	0	Buck's extension	5 wks.	5 wks.	1 day	0	0	Cast, no shortening, no limitation of motion	Complete recovery.
57	J. S.	12 yrs.	M	7-16-09	Middle of shaft	0	Buck's extension	6 wks.	6 wks.	1 wk.	2 wks.	0	Crutches, no shortening	Complete recovery.
58	L. G.	7 yrs.	M	7-29-12	Lower ⅓ of shaft of femur	0	Buck's extension	9 wks.	8.5 wks.	2 days	0	0	Much weakness, no shortening	Complete recovery.
59	H. K.	4 yrs.	F	1-28-12	Middle ⅓ of femur	0	Buck's extension	10 wks.	10 wks.	9 wks.	0	0	Not walking, no shortening	Complete recovery.
60	J. L.	5 yrs.	M	9-3-10	Lower ⅓ of right femoral shaft	0	Buck's extension	7 wks.	7 wks.	0	0	0	Good, no shortening	Complete recovery.

61	J. K.	9 yrs.	M	6-1-11	Juncture of upper and middle ⅓	½ in.	Buck's extension	8 wks.	8 wks.	0	0	0	Weakness, no shortening	Complete recovery.
62	L. P.	5 yrs.	M	6-10-13	Oblique fracture lower ⅓ shaft of right femur	1½ in.	Buck's extension	7 wks.	7 wks.	3 days	Cast 1 wk.	0	Cast, ½ in. shortening	Complete recovery, slight lameness.
63	H. W.	4 yrs.	M	8-26-13	Middle of shaft of left femur	0	Buck's extension	7 wks.	7 wks.	0	0	0	No shortening, could not walk well	Complete recovery.
64	G. K.	12 yrs.	M	12-17-09	Lower ⅓ right femoral shaft	0	Buck's extension	5.5 wks.	5.5 wks.	2 days	3 mos.	0	Crutches; plaster cast (4 wks.)	Complete recovery.
65	E. T.	5 yrs.	M	8-15-12	Middle	0	Buck's extension	7 wks.	7 wks.	0	0	0	Unable to walk. No shortening	Complete recovery.
66	L. W.	7 yrs.	M	10-15-10	Lower ⅓ right femur	0	Long axis extension, short interior and posterior splint	3 wks.	5.5 wks.	7 days	3 mos. Cast 3 wks.	0	Crutches	Complete recovery, except pain damp days.
67	J. G.	11 yrs.	M	9-11-06	Upper ⅓ right femur	¾–1 in.	Buck's extension	2.5 mos.	2.5 mos.	6 days	1 mo.	0	Walks well on crutch ½ in. shortening	Complete recovery.
68	M. J.	7 yrs.	F	3-7-13	Left femur, supracondylar	0	Buck's extension	9 wks.	9 wks.	0	3 mos.	0	Crutches, ½ in shortening, limp	Complete recovery.
69	R. D.	3 yrs.	M	6-5-13	Middle of left femur	0	Buck's extension	7 wks.	7 wks.	0	0	0	Good. No shortening, some weakness	Complete recovery.
70	A. F.	7 yrs.	M	8-9-08	Lower ⅓ of shaft of left femur	0	Buck's extension	12 wks.	12 wks.	2 days	6 mos.	0	Plaster cast and crutches, no shortening	Complete recovery.
71	E. B.	5 yrs.	M	9-5-09	Upper ⅓ of shaft of right femur	0	Buck's extension	7 wks.	7 wks.	2 days	0	0	Walked. No cast. Some limp	Complete recovery.
72	M. H.	5 yrs.	M	11-27-11	Upper ⅓ of shaft of left femur	0	Buck's extension	11 wks.	11 wks.	2 days	0	0	Good. No shortening.	Complete recovery.
73	J. L.	6 yrs.	M	11-30-12	Lower ⅓ of shaft of right femur	0	Buck's extension	13 wks.	13 wks.	0	0	0	Slight limp. No shortening	Complete recovery.
74	I. R.	6 yrs.	M	6-8-13	Oblique fracture in middle ⅓ left femur	¼ in.	Buck's extension and cast	6 wks.	6 wks.	2 days	2 wks.	0	Crutches. ¼ in. shortening	Complete recovery.
75	J. L.	6 yrs.	F	5-21-06	Middle ⅓	0	Buck's extension	5.5 wks.	5.5 wks.	0	0	0	Walked with slight limp	Complete recovery.
76	W. S.	11 yrs.	M	12-31-13	Shaft right femur, overlapping upper ⅓	0	Open operation, plates	12 wks.	12 wks.	0	10 days	0	Considerable limp. No shortening. Incision looks well	Complete recovery.
77	J. M.	11 yrs.	M	8-17-06	Upper ⅓ of shaft of right femur	2 in. ununited	Cast. Operated 3 times. Fragments wired	12 wks.	1 yr.	1 wk.	3 mos.	0	Ununited.	½ in. shortening knee stiff, leg crooked, no pain.
78	J. H.	12 yrs.	M	10-23-11	Just below middle of left femur	2½ in.	Buck's extension, open, and plates	6 mos.	6 mos.	1 mo.	0	0	Walks with limp	1¼ in. shortening, slight limp, no pain, runs well.
79	H. R.	12 yrs.	M	8-30-10	Middle of femur	½ in.	Buck's extension	6 wks.	6 wks.	1 day	1 mo.	10 days	Crutches, ½ in. shortening	½ in. shortening, stiffness in knee, no pain.

CHART I. END-RESULTS IN SIMPLE FRACTURE OF FEMORAL SHAFT—*Continued*

No.	Name	Age	Sex	Date of Fracture	Location	Primary Shortening	Treatment	Duration of Extension	Bed	Chair	Crutch	Cane	Condition at Discharge	Present Condition
80	J. D.	7 yrs.	M	9-12-13	Upper ⅓ of shaft of right femur	0	Buck's extension	8 wks.	8 wks.	1 day	0	0	No shortening, no limitation of motion, no pain	¼ in. shortening, pain in wet weather.
81	J. S.	8 yrs.	M	5-19-07	Middle ⅓ of femur	¼ in.	Buck's extension	7.5 wks.	7.5 wks.	3 days	2 wks.	0	Crutches, ¼ in. shortening	¼ in. shortening, otherwise complete recovery.
82	J. H.	10 yrs.	M	5-2-07	Lower ⅓	¼ in.	Buck's extension	6.5 wks.	6.5 wks.	3 days	1 mo.	1 wk.	Crutches, ¼ in. shortening	¼ in. shortening, otherwise complete recovery.
83	J. P.	4 yrs.	M	5-1-10	Middle of right shaft	¼ in.	Buck's extension	5 wks.	5 wks.	0	0	0	¼ in. shortening, no lameness	¼ in. shortening, otherwise complete recovery.
84	J. R.	10 yrs.	M	11-21-12	Upper ⅓ of shaft of left femur	0	Buck's extension	6 wks.	6 wks.	1 wk.	0	0	No shortening, slight stiffness in knee	1½ ins. shortening, some stiffness in knee, limp.
85	J. G.	6 yrs.	M	8-25-13	Middle of right femur	½ in.	Plaster cast	0	9 wks.	0	0	0	No cast, ½ in. shortening, limp	½ in. shortening, otherwise complete recovery.
86	H. D.	5 yrs.	F	10-10-10	Middle ⅓ of left femur	0	Buck's extension	8 wks.	8 wks.	0	0	0	Braces for both legs to overcome angulation	Braces, no shortening, some angulation.
87	M. E.	20 yrs.	F	1-15-11	Middle ⅓	⅜ in.	Buck's extension and plaster cast	7 wks. and plaster cast 3 wks. more	7 wks.	2 days	0	2 mos.	⅜ in. shortening, knee very stiff	Complete recovery.
88	J. E.	15 yrs.	M	12-15-13	Middle of shaft of right femur	0	Buck's extension	Open operation, plates	17 wks.	2 days	5 wks.	0	No shortening, no limitation of motion	Complete recovery.
89	J. F.	17 yrs.	M	10-24-07	Upper ⅓ of right femur	¼ in.	Buck's extension	12 wks.	12 wks.	1 wk.	2 wks.	0	Crutches, ¼ in. shortening	Complete recovery.
90	F. E.	13 yrs.	M	7-18-07	Middle	0	Buck's extension	8 wks.	8 wks.	0	0	0	Good. No shortening, stiffness in knee	Complete recovery.
91	J. P.	13 yrs.	F	5-28-08	Lower ⅓ of right shaft	0	Buck's extension	9 wks.	9 wks.	1 day	1 mo.	0	Crutches, no shortening	Complete recovery.
92	J. G.	16 yrs.	M	3-7-06	Middle of femur	0	Buck's extension	7 wks.	7 wks.	4 days	2.5 mos.	2 wks.	Crutches, no shortening, stiffness in knee	Complete recovery, slight limp.
93	F. R.	17 yrs.	F	11-18-11	Middle of shaft	0	Buck's extension	5 wks.	5 wks.	2 days	0	0	No crutches, no shortening, weakness	Complete recovery.
94	M. R.	15 yrs.	F	11-18-09	Lower ⅓ of shaft of right femur	¼ in.	Buck's extension	8 wks.	8 wks.	2 days	1 mo.	0	Crutch, stiffness in knee, ¼ in. shortening	¼ in. shortening, no limitation of motion.
95	T. W.	16 yrs.	M	12-3-10	Middle ⅓	½ in.	Buck's extension	15 wks.	15 wks.	1 wk.	1 mo.	0	Crutches, ½ in. shortening	½ in. shortening, no limitation of motion, no pain.
96	J. F.	15 yrs.	M	10-20-07	Middle of shaft	1 in.	Buck's extension	7.5 wks.	7.5 wks.	3 days	6 wks.	1 mo.	Crutches, 1 in. shortening	1 in. shortening, no limitation of motion, limp.
97	M. G.	23 yrs.	M	4-17-12	Supracondylar	1 in.	Buck's extension	7 wks.	7 wks.	1 wk.	3 mos.	3 mos.	Crutches, 1 in. shortening, stiffness in knee	1 in. shortening, no limitation of motion.
98	P. W.	22 yrs.	M	3-2-08	Shaft of femur, supracondylar	¼ in.	Buck's extension	10 wks.	10 wks.	1 wk.	3 mos.	1 mo.	¼ in. shortening, stiffness in knee	¼ in. shortening, no limitation of motion.

99	B. L.	24 yrs.	F	12-27-10	Middle	1½ in.	Buck's extension	6 mos.	6 mos.	2 wks.	ever	since	1½ in. shortening, stiffness in knee	1½ in. shortening.
100	A. F.	21 yrs.	F	3-18-12	Lower ⅓	½ in.	Buck's extension	7 wks.	7 wks.	1 wk.	3 mos.	2 mos.	Crutches, ½ in. shortening, stiffness in knee	½ in. shortening, no limitation of motion.
101	J.G.B.	19 yrs.	M	5-20-12	Middle of right femur	2 in.	Buck's extension	11 wks.	11 wks.	2 days	2 mos.	1 mo.	Crutches, 2 ins. shortening, stiffness in knee	2 ins. shortening, angulation, no limitation of motion.
102	F. C.	18 yrs.	M	9-8-05	Upper ⅓ of shaft of femur	⅜ in.	Buck's extension	7 wks.	7 wks	5 days	2 mos.	1 mo.	Crutches, ½ in. shortening, stiffness in knee	½ in. shortening, otherwise complete recovery.
103	J. B.	13 yrs.	M	10-10-05	Middle of shaft	⅜ in	Buck's extension	9 wks.	9 wks.	2 days	1 mo.	2 wks.	Crutches, ⅜ in. shortening, stiffness in knee	½ in. shortening, otherwise complete recovery.
104	J. K.	15 yrs.	M	1-5-07	Shaft left femur	1 in.	Buck's extension	8 wks.	8 wks.	2 days	4 mos.	3 wks.	Crutches, 1 in. shortening, stiffness in knee	Complete recovery except 1 in. shortening.
105	J. C.	17 yrs.	M	6-8-12	Lower ⅓ of shaft of right femur	½ in.	Buck's extension	8 wks.	8 wks.	2 days	6 wks.	2 wks.	Crutches, ½ in. shortening, stiffness in knee	No pain nor limitation of motion.
106	P. T.	30 yrs.	M	11-8-11	Middle of femur	0	Buck's extension	8 wks.	8 wks.	2 days	3 mos.	1 wk.	No shortening, slight stiffness in knee	Complete recovery, except slight exostosis.
107	B. S.	27 yrs.	M	8-11-06	Middle of shaft	0	Buck's extension	7.5 wks.	7.5 wks.	3 days	2.5 mos.	2 mos.	No shortening, slight stiffness in knee	Complete recovery, except slight limp.
108	S. M.	30 yrs.	F	3-27-12	Lower ⅓ of femur	0	Buck's extension	6 wks.	6 wks.	1 wk.	2 mos.	2 wks.	After return home, went to bed. Slight stiffness in knee	Complete recovery.
109	U. D.	29 yrs.	M	7-19-05	Supracondylar	0	Buck's extension	8 wks.	8 wks.	1 wk.	2 mos.	1 mo.	No shortening, slight stiffness in knee	Complete recovery.
110	W. S.	30 yrs.	M	7-15-08	Upper and middle ⅓ of shaft of right femur	0	Buck's extension	6 wks.	6 wks.	2 wks.	3 mos.	1 mo.	Crutches, no shortening, stiffness in knee	Complete recovery.
111	W. H.	38 yrs.	M	10-11-12	Shaft of right femur, supracondylar	0	Buck's extension	7 wks.	7 wks.	1 wk.	5 wks.	4 wks.	Crutches, stiffness in knee	Complete recovery, except slight limp.
112	F. Q.	34 yrs.	M	11-27-13	Subtrochanteric	½ in.	Buck's extension	16 wks.	16 wks.	1 wk.	0	6 mos.	Went back to bed. 2 ins. shortening, stiffness in knee	½ in. shortening, very slight limp.
113	A. P.	26 yrs.	M	7-22-13	Middle	½ in.	Buck's extension	7 wks.	7 wks.	1 wk.	1 mo.	1 mo.	½ in. shortening, stiffness in knee	½ in. shortening, no pain, no stiffness.
114	M. L.	38 yrs.	M	5-16-08	Lower ⅓ right femur	2½ ins.	Buck's extension	7 wks.	8 wks.	1 wk.	3 mos.	Ever since	Crutches, 2½ ins. shortening, stiffness in knee	2½ ins. shortening, brace, limp, no pain.
115	W. S.	32 yrs.	M	2-27-14	Upper ⅓ of left femur	¼ in.	Buck's extension	6 wks.	6 wks.	2 days	Ever since	0	Stiffness in knee, ¼ in. shortening	No stiffness, ¼ in. shortening.
116	J. A.	38 yrs.	M	2-20-13	Lower ⅓ right femur	½ in.	Buck's extension	8 wks.	8 wks.	3 days	6 mos.	4 mos.	Crutches, ½ in. shortening, stiffness in knee	½ in. shortening, pain in damp weather.
117	W. H.	30 yrs.	M	6-11-07	Upper ⅓ of femoral shaft	¼ in.	Buck's extension	7 wks.	7 wks.	1 wk.	4 mos.	2 mos.	Crutches, ¼ in. shortening, stiffness in knee	¼ in. shortening, no limitation of motion, no limp.
118	J.R.C.	30 yrs.	M	10-2-14	Middle of femur	½ in.	Buck's extension	14 wks.	14 wks.	1 wk.	3 mos.	3 mos.	Crutches, ½ in. shortening, stiffness in knee	No limitation of motion, ½ in. shortening.
119	M. L.	26 yrs.	M	4-8-11	Middle of shaft of right femur	¼ in.	Buck's extension	5 wks.	5 wks.	1 day	2 mos.	1 mo.	¼ in. shortening, stiffness in knee	¼ in. shortening, no limitation of motion, no limp.
120	N. S.	27 yrs.	F	2-16-07	Supracondylar, exterior condyle involving joint	1¼ in.	Buck's extension	7 wks.	7 wks.	1 wk.	3.5 mos.	2 mos.	1¼ ins. shortening, stiffness in knee	1¼ ins. shortening, pain on damp days.
121	J. W.	35 yrs.	M	3-3-10	Lower ⅓ of shaft of femur	2 in.	Buck's extension	14 wks.	14 wks.	1 wk.	3 mos.	3 mos.	Crutches, 2 ins. shortening, stiffness in knee	No limitation of motion, 2 ins. shortening.

CHART I. END-RESULTS IN SIMPLE FRACTURE OF FEMORAL SHAFT—*Continued*

No.	Name	Age	Sex	Date of Fracture	Location	Primary Shortening	Treatment	Duration of Extension	Bed	Chair	Crutch	Cane	Condition at Discharge	Present Condition
122	J. S.	35 yrs.	M	8-13-12	Middle of shaft of right femur	0	Buck's extension	12 wks.	12 wks.	1 wk.	3 mos.	2 mos.	½ in. shortening, slight stiffness in knee	½ in. shortening, no limitation of motion.
123	F. M.	30 yrs.	M	10-29-11	Lower ⅓ of shaft of femur	1 in.	Buck's extension	7 wks.	7 wks.	2 days	3 mos.	1 mo.	Crutches, 1 in. shortening, slight stiffness in knee	1 in. shortening, pain in damp weather.
124	M K.	38 yrs.	M	3-16-12	Shaft right femur, supracondylar	½ in.	Buck's extension	7 wks.	7 wks.	2 days	4 mos.	2 mos.	Crutches, ½ in. shortening, stiffness in knee	½ in. shortening, wears high heel.
125	R. F.	23 yrs.	M	7-17-11	Left middle	¾ in.	Lane plate, open operation, plaster cast	0	12 wks.	1 wk.	1 mo.	0	Cast, crutches, ¾ in. shortening, stiffness in knee	¾ in. shortening, no limitation of motion.
126	J. H.	38 yrs.	M	10-18-09	Lower ⅓ right femur	1 in.	Buck's extension	6 wks.	6 wks.	1 wk.	1 mo.	2 wks.	Right knee very stiff, 1 in. shortening	1 in. shortening, no limitation of motion, limp.
127	P.McG.	38 yrs.	M	7-20-05	Irregular transverse fracture lower ⅓ of left femur	½ in.	Buck's extension	7 wks.	7 wks.	2 days	6 mos.	2 mos.	Crutches, ½ in. shortening, stiffness in knee	½ in. shortening, pain in wet weather.
128	E. F.	35 yrs.	F	5-29-13	Middle of femoral shaft	1 in.	Plated, plaster cast	0	11 wks.	2 days	6 mos.	3 mos.	Crutches, 1 in. shortening, stiffness in knee	1 in. shortening, slight limp, slightly stiff.
129	R. L.	53 yrs.	M	10-1-05	Middle of femur	0	Buck's extension	9 wks.	9 wks.	1 wk.	3 mos.	2 mos.	Crutches, much stiffness in knee, no shortening.	Complete recovery.
130	G. M.	54 yrs.	M	5-10-06	Upper ⅓	0	Buck's extension	8 wks.	8 wks.	1 wk.	4 mos.	1 mo.	No shortening, stiffness in knee	Complete recovery.
131	M. S.	50 yrs.	F	12-2-10	Middle of shaft of right femur	½ in.	Buck's extension	6 mos.	6 mos.	2 days	3 yrs.	0	Went home in ambulance. Right knee swollen and stiff	Leg rebroken at knee 7 mos. ago. No shortening.
132	W.F.R.	50 yrs.	M	9-10-06	Upper ⅓ of shaft	1½ in.	Buck's extension	6 wks.	3.5 mos.	1 wk.	6 mos.	2 mos.	Crutches, stiffness in knee, 1½ ins. shortening	1½ ins. shortening, some pain in wet weather.
133	J. G.	40 yrs.	M	9-13-11	Supracondylar	3½ in.	Buck's extension. Brace from waist to heel	5 mos.	5 mos.	0	7 mos.	2 yrs.	3¼ ins. shortening, stiffness in knee	3½ ins. shortening, limitation of motion, pain wet weather.
134	A. W.	56 yrs.	F	3-27-13	Lower ⅓	2 in.	Buck's extension	5 wks.	5 wks.	1 wk.	Ever	Since	2 ins. shortening, stiffness in knee	2 ins. shortening, crutch, lame in wet weather.
135	D.McF.	52 yrs.	M	11-29-10	Middle ⅓ of shaft of femur	2½ in.	Buck's extension	4.5 mos.	4.5 mos.	2 wks.	1 yr.	Ever since	Crutches, much limitation of motion, 2¾ ins. shortening	2½ ins. shortening, ulcer tip of heel, stiffness.
136	D.McL.	52 yrs.	M	6-28-12	Middle of right femur	¾ in.	Buck's extension	9 wks.	9 wks.	5 days	5 mos.	2 mos.	¾ in. shortening, stiffness in knee, went to bed (home)	¾ in. shortening, stiffness in knee.
137	A. R.	60 yrs.	F	9-9-07	Upper ⅓	1½ in.	Buck's extension	8 wks.	8 wks.	1 wk.	6 mos.	1 mo.	Crutches, stiffness in knee	1½ ins. shortening, no pain.

138	G. D.	58 yrs.	M	11-4-06	Middle ⅓ of left femur	1¾ in.	Buck's extension	12 wks.	12 wks.	1 wk.	8 mos.	Ever since	Much stiffness in knee, 1¾ ins. shortening	1¾ ins. shortening, stiffness, pain in wet weather.
139	J. B.	57 yrs.	M	3-16-10	Middle of shaft of right femur	1½ in.	Buck's extension	7 wks.	7 wks.	5 days	6 mos.	1.5 yrs.	Crutches, 1½ ins. shortening, stiffness in leg and knee	1½ ins. shortening, does not use cane.
140	T. S.	52 yrs.	M	6-4-09	Middle of shaft of left femur	1½ in.	Buck's extension	7 wks.	7 wks.	1 wk.	5 mos.	1 mo.	1½ ins. shortening, much stiffness in knee	1½ ins. shortening, no stiffness, no pain, limp.
141	A. W.	58 yrs.	F	1-21-13	Left femur, subtrochanteric	1½ in.	Buck's extension	8 wks.	8 wks.	1 day	3 wks.	10 mos.	Crutches, 1½ ins. shortening, stiffness in knee	1½ ins. shortening, slightly stiff, pain wet weather.
142	E. J.	50 yrs.	M	1-20-11	Lower ⅓ right femur	1 in.	Buck's extension	7 wks.	7 wks.	2 days	3 mos.	3 mos.	Crutches, 1 in. shortening, stiffness in knee	1 in. shortening, no limitation of motion, limp.
143	G. D.	53 yrs.	M	11-4-06	Middle of femoral shaft	¾ in.	Buck's extension	11 wks.	11 wks.	1 wk.	5 mos.	3 mos.	Crutches, ¾ in. shortening, stiffness in knee.	¾ in. shortening, no limitation of motion, limp.
144	V. S.	57 yrs.	F	9-16-11	Upper ⅓ of left femur	1½ in.	Buck's extension	8 wks.	8 wks.	1 wk.	0	Ever since	1½ ins. shortening, much stiffness in knee	1½ ins. shortening, no stiffness, uses cane.
145	A. S.	56 yrs.	F	11-20-13	Upper ⅓ of shaft of left femur	2¼ in.	Buck's extension	9 wks.	7 mos.	1 wk.	Ever	Since	Went home in ambulance, 2¼ ins. shortening, stiffness	2¼ ins. shortening, pain in wet weather.
146	A. J.	54 yrs.	F	4-30-08	Middle of shaft of femur	1½ in.	Buck's extension	8 wks.	8 wks.	1 wk.	6 mos.	2 mos.	Crutches, 1½ ins. shortening, stiffness in knee	1½ ins. shortening, wears high heel, stiffness.
147	A. L.	60 yrs.	F	2-15-11	Middle of left femur	2 in.	Buck's extension	11 wks.	11 wks.	1 wk.	Ever since		Crutches, 2 ins. shortening, stiffness in knee	2 ins. shortening, stiffness, pain wet weather.
148	W. V.	60 yrs.	M	5-16-08	Upper ⅓ of left shaft	½ in.	Buck's extension	6.5 wks.	6.5 wks.	4 days	6 mos.	1 mo.	½ in. shortening, stiffness in knee	½ in. shortening, limp, no pain.
149	W. K.	50 yrs.	M	1-7-12	Lower ⅓ of shaft of femur	¾ in.	Buck's extension	5 wks.	5 wks.	1 wk.	4.5 mos.	4 mos.	¾ in. shortening, stiffness in knee	¾ in. shortening, no limitation of motion.
150	M. W.	60 yrs.	F	3-2-07	Lower ⅓ right femur	½ in.	Buck's extension	5.5 wks.	5.5 wks.	2 days	6 mos.	1 mo.	½ in. shortening, stiffness in knee	½ in. shortening, slight limp.
151	G.R.R.	50 yrs.	M	9-7-10	Middle of femur	1¼ in.	Buck's extension	7.5 wks.	7.5 wks.	4 days	3 mos.	4 mos.	1¼ in. shortening, stiffness in knee	1¼ in. shortening, pain in wet weather.
152	S. K.	52 yrs.	M	10-27-13	Middle of shaft of left femur	1 in.	Buck's extension	7 wks.	7 wks.	2 days	4 mos.	2 mos.	Crutches, 1 in. shortening.	1 in. shortening, pain in wet weather.
153	J. B.	49 yrs.	F	12-21-13	Middle of femur	½ in.	Buck's extension	6 wks.	6 wks.	2 days	Ever since	0	Crutches, 1 in. shortening, stiffness in knee	1 in. shortening, crutches, no stiffness.
154	J.McL.	55 yrs.	M	2-9-10	Middle of left femur	1½ in.	Buck's extension	11 wks.	11 wks.	1 wk.	4.5 mos.	2 mos.	1½ in. shortening, stiffness in knee	1½ in. shortening, no limitation of motion.
155	A. M.	56 yrs.	F	4-5-13	Right supracondylar	½ in.	Buck's extension	9 wks.	9 wks.	1 wk.	2 mos.	3 mos.	Crutches, ½ in. shortening, stiffness in knee	½ in. shortening.
156	W. M.	53 yrs.	M	8-2-09	Lower ⅓ left femur	1½ in.	Buck's extension	13 wks.	13 wks.	1 wk.	Ever since	0	1½ in. shortening, stiffness in knee	1¼ in. shortening, crutches.
157	A. C.	50 yrs.	F	6-14-13	Just below greater trochanter. Lower fragment pulled in left femur	Shortening due to previous injury	Buck's extension	4 wks.	4 wks.	1 wk.	4 mos.	6 wks.	Plaster cast, much stiffness in knee	Limp due to previous injury.
158	W. M.	49 yrs.	M	12-4-04	Middle and lower ⅓ of right femur	½ in.	Buck's extension	7 wks.	7 wks.	1 wk.	5 mos.	3 mos.	½ in. shortening, much stiffness in knee	½ in. shortening, no limitation of motion.

CHART I. END-RESULTS IN SIMPLE FRACTURE OF FEMORAL SHAFT—*Continued*

No.	Name	Age	Sex	Date of Fracture	Location	Primary Shortening	Treatment	Duration of Extension	Bed	Chair	Crutch	Cane	Condition at Discharge	Present Condition
159	W.McC.	45 yrs.	M	2-9-12	Ununited fracture of lower ⅓ of right femur	1¼ in.	Ununited fracture operated twice, then plated. Infection	0	3 wks.	12 wks.	9 mos.	9 mos.	Crutches, stiffness in knee	Incision breaks down every 8 wks.
160	L. P.	45 yrs.	F	5-21-07	Middle of right femur	2 in.	Buck's extension	7 wks.	7 wks.	2 wks.	1 yr.	4 yrs.	Crutches, stiffness in knee	2 ins. shortening, stiffness pain damp weather.
161	L. W.	45 yrs.	M	7-26-07	Middle of right femur	1 in.	Buck's extension	6 wks.	6 wks.	1 day	3 mos.	1 mo.	1 in. shortening, limitation of motion, stiffness in knee	1 in. shortening, stiffness, pain damp weather.
162	W.H.B.	43 yrs.	M	2-12-12	Lower ⅓	1 in.	Buck's extension	11 wks.	11 wks.	1 wk.	2 mos.	1 mo.	Crutches, 1 in. shortening, stiffness in knee	1 in. shortening pain on, damp days.
163	W. W.	48 yrs.	M	9-8-06	Middle and upper ⅓ of right femur	1¼ in.	Buck's extension	10 wks.	10 wks.	3 days	6 mos.	2 mos.	Crutches, 1¼ in. shortening, much limitation of motion (knee)	1¼ in. shortening, fairly good motion.
164	M.O'C.	42 yrs.	F	10-10-11	Middle of shaft of left femur	2 in.	Buck's extension	8 wks.	8 wks.	2 days	6 mos.	1 mo.	Crutches, 2 ins. shortening, limitation of motion (knee)	2 ins. shortening. Wears high heel.
165	B. Q.	47 yrs.	M	8-20-12	Middle ⅓ of right femur	1¾ in.	Buck's extension and cast	Extension 6 wks. Cast 3 mos.	3 mos.	6 mos.	7 mos.	Ever since	1¾ in. shortening, crutches, stiffness in knee	1¾ in. shortening, stiffness in knee.
166	L. P.	40 yrs.	M	11-14-07	Upper ⅓ of shaft of femur	¾ in.	Buck's extension	7 wks.	7 wks.	1 wk.	4 mos.	2 mos.	Crutches, stiffness in knee	¾ in. shortening, limp.
167	H.McF.	46 yrs.	M	3-10-08	Juncture of upper and middle ⅓ of left femur	1½ in.	Buck's extension	7 wks.	7 wks.	1 wk.	4 mos.	1 mo.	Crutches, stiffness in knee, 1½ in. shortening.	1½ in. shortening, limp.
168	J. K.	42 yrs.	M	1-18-10	Lower ⅓ left femur	1½ in.	Buck's extension	13 wks.	13 wks.	1 wk.	8 mos.	2 mos.	Crutches, 1½ in. shortening, stiffness in knee	1½ in. shortening, limp.
169	J. D.	48 yrs.	M	12-8-07	Middle and lower ⅓ of right femur	1 in.	Buck's extension	7 wks.	7 wks.	1 wk.	3.5 mos.	2 wks.	Crutches, 1 in. shortening, stiffness in knee	1 in. shortening, slight limp.
170	F. C.	41 yrs.	M	6-3-06	Middle	¼ in.	Buck's extension	7 wks.	7 wks.	1 wk.	3 mos.	2 mos.	Crutches, ¼ in. shortening, stiffness in knee	¼ in. shortening, slight pain in wet weather.
171	J. M.	43 yrs.	M	11-22-06	Femoral shaft	1 in.	Buck's extension	8 wks.	8 wks.	5 days	3 mos.	6 mos.	Crutches, 1 in. shortening, stiffness in knee	1 in. shortening, slight limp.
172	W.O'M.	40 yrs.	M	1-10-13	Lower ⅓ left femur	1 in.	Buck's extension	12 wks.	12 wks.	1 wk.	2 mos.	4 mos.	Crutches, 1 in. shortening, stiffness in knee	1 in. shortening, stiffness in knee, limp.
173	T. B.	48 yrs.	M	8-9-12	Middle of shaft of femur	2 in.	Buck's extension	8 wks.	7 wks.	1 wk.	6 mos.	Ever since	Crutches, 2 ins. shortening, stiffness in knee	2 ins. shortening, stiffness in knee, limp.
174	S. R.	43 yrs.	M	3-19-06	Middle of shaft of right femur	¾ in.	Buck's extension	7 wks.	7 wks.	3 days	2 mos.	1 mo.	¾ in. shortening, crutches, stiffness in knee	¾ in. shortening.
175	W. B.	42 yrs.	M	9-17-06	Middle of shaft of femur	1 in.	Buck's extension	9 wks.	9 wks.	1 wk.	4 mos.	2 mos.	1 in. shortening, stiffness in knee	1 in. shortening, pain in wet weather.
176	E. R.	42 yrs.	F	3-22-13	Upper ⅓ of femur	0	Buck's extension	16 wks.	Ever since	0	0	0	Ununited. Taken home in ambulance	1 in. shortening, fibrous union.

177	L. D.	65 yrs.	M	8-3-12	Middle of femur	1 in.	Buck's extension	12 wks.	3 mos.	1 wk.	5 mos.	Still	1 in. shortening, stiffness in knee	1 in. shortening, no stiffness.
178	J. M.	65 yrs.	M	11-26-13	Upper ⅓	2½ in.	Sand bags, Buck's extension	5 wks. 1 wk.	8 wks.	1 wk.	3 mos.	4 mos.	2½ ins. shortening. Went to bed at home	2½ ins. shortening, stiffness. Uses cane.
179	L. K.	67 yrs.	F	6-14-11	Upper ⅓ right femur	½ in.	Buck's extension	12 wks.	3 mos.	1 wk.	Ever since	0	Crutches, ½ in. shortening, stiffness in knee	Result good, unable to move for rheumatism.
180	W. M.	65 yrs.	M	12-14-07	Supracondylar	2¾ in.	Buck's extension	8 wks.	8 wks.	1 wk.	9 mos.	3 mos.	2¾ ins. shortening, stiffness in knee	2¾ ins. shortening, pain in wet weather, limp.
181	J. D.	65 yrs.	M	4-7-05	Supracondylar	¾ in.	Buck's extension	9 wks.	9 wks.	1 wk.	5 mos.	4 mos.	¾ in. shortening, crutches, much stiffness in knee	¾ in. shortening, slight limp.
182	M. W.	63 yrs.	F	3-27-07	Supracondylar	1¾ in.	Buck's extension	8 wks.	8 wks.	1 wk.	6 mos.	4 mos.	1¾ in. shortening, stiffness in knee	1¾ in. shortening, pain in wet weather, limp,
183	H. C.	62 yrs.	F	4-2-05	Supracondylar	0	Buck's extension	8 wks.	8 wks.	0	0	0	Went home in ambulance, stiffness in knee	Rheumatic arthritis knees, union good.
184	E. K.	61 yrs.	F	12-8-12	Lower ⅓ right femur	2 in.	Wired	0	29 wks.	1 wk.	Ever since	0	Crutches, 2 ins. shortening, stiffness in keen	2 ins. shortening, no stiffness.
185	M. R.	61 yrs.	M	3-27-11	Middle left femur	1 in.	Buck's extension	6 wks.	6 wks.	1 wk.	2 mos.	1 mo.	1 in. shortening, stiffness in knee	1 in. shortening, no pain or stiffness.
186	A. A.	68 yrs.	F	2-5-08	Middle ⅓ right femur	1½ in.	Buck's extension	16 wks.	16 wks.	1 wk.	4 mos.	2 mos.	1½ in. shortening, stiffness in knee	1½ in. shortening slight stiffness, no pain.
187	C. P.	65 yrs.	M	8-31-10	Upper ⅓ right femur	1½ in.	Buck's extension	7 wks.	7 wks.	1 wk.	7 mos.	Ever since	Union not firm, 1½ in. shortening	Firm union, 1½ in. shortening, stiffness knee.
188	A.O'D.	65 yrs.	F	1-26-12	Middle of femur	0	Buck's extension	5 mos.	5 mos.	0	0	0	Ununited fracture	Ununited.
189	S. M.	64 yrs.	F	12-6-13	Upper ⅓ of shaft of right femur	2 in.	Buck's extension	7 wks.	7 wks.	1 wk.	4 mos.	1 mo.	2 ins. shortening, much stiffness in knee	2 ins. shortening, no pain. slight stiffness (knee).
190	G. W.	64 yrs.	M	2-15-11	Supracondylar, left femur	1 in.	Buck's extension	13 wks.	13 wks.	1 wk.	Ever	Since	Crutches, stiffness in knee	1 in. shortening, pain in wet weather.
191	C. B.	65 yrs.	F	9-9-13	Lower ⅓ of shaft of right femur	1 in.	Buck's extension	12 wks.	12 wks.	1 wk.	Ever since	0	Crutches, 1 in. shortening, stiffness in knee	Crutches, 1 in. shortening.
192	M. N.	70 yrs.	F	6-10-13	Upper ⅓ left femoral shaft	0	Buck's extension	11 wks.	11 wks.	1 wk.	2 days	2 wks.	Crutches, no shortening, stiffness in knee	Complete recovery, pain on damp days.
193	H. W.	80 yrs.	F	7-22-08	Upper ⅓ left femur	¾ in.	Buck's extension		10 wks.	1 wk.	6 mos.	Ever since	¾ in. shortening, slight stiffness in left knee	¾ in. shortening, stiffness left knee, limp.
194	R.H.R.	74 yrs.	M	10-1-10	Middle of shaft	¾ in.	Buck's extension	8 wks.	8 wks.	1 wk.	4 mos.	6 mos.	¾ in. shortening, stiffness in knee	¾ in. shortening, no pain, stiffness in knee.
195	J. K.	70 yrs.	M	8-18-09	Upper ⅓ left femur	1½ in.	Buck's extension		5 wks.		6 mos.	1 yr.	1 in. shortening, stiffness in knee on affected side	1 in. shortening, gets around well.
196	A. K.	70 yrs.	F	11-15-04	Middle of shaft of femur	¾ in.	Buck's extension	10 wks.	10 wks.	1 wk.	4 mos.	4 mos.	¾ in. shortening, stiffness in knee	¾ in. shortening, otherwise complete recovery.
197	C. C.	72 yrs.	F	10-1-08	Middle right femur	½ in.	Buck's extension	6 wks.	6 wks.	1 wk.	6 mos.	0	Crutches, ½ in. shortening	In bed with rheumatism, ½ in. shortening.
198	B. M.	70 yrs.	M	7-17-08	Lower ⅓ right femur, supracondylar	½ in.	Buck's extension	5 wks.	5 wks.	1 day	1 mo.	Ever since	Crutches, ½ in. shortening	½ in. shortening, stiffness due to weakness.

No.	Name	Age	Sex	Date of Fracture	Location	Treatment	Deaths
199	M. B.	5 years	F	8–4–10	Middle of right femur	Buck's extension	Died in 3 hours from shock.
200	J. C.	2 years	M	3–7–11	Middle of left femur	Buck's extension overhead	Died in 2 months from acute gastro-enteritis.
201	P. McC.	3 years	M	7–18–11	Middle of right femur	Buck's extension	Died in 4½ weeks from tuberculosis.
202	K. R.	35 years	F	9–7–07	Middle of left shaft	Buck's extension	Died in 3 weeks from pneumonia.
203	S. S.	24 years	M	9–5–12	Upper ⅓ of left femur	Buck's extension	Died in 18 days from lobar pneumonia.
204	J. McM.	32 years	M	12–2–10	Upper ⅓ right femur	Buck's extension	Died in 3 days from delirium tremens.
205	G. W.	20 years	M	9–8–09	Middle of left femur	In bed. Sand bags	Died in 2 hours from shock.
206	B. M.	35 years	M	8–10–11	Middle of left femur	Buck's extension	Died in 12 wks. from inte'al injuries and shock.
207	J. G.	47 years	M	7–5–12	Middle of left femur	Buck's extension	Died in 6 hours from shock.
208	C. L.	48 years	M	2–4–04	Left femur, subtrochanteric	Buck's extension	Died in 12 hours from shock.
209	B. C.	54 years	M	5–11–09	Upper ⅓ left femur	Buck's extension	Died in 2 days from delirium tremens.
210	P. C.	52 years	M	5–18–06	Upper ⅓ right femur	Buck's extension	Died in 9 days from delirium tremens.
211	M. F.	40 years	M	11–5–07	Right femoral shaft, supra-condylar	Buck's extension	Died in 8 weeks from delirium tremens.
212	J. T.	56 years	M	4–12–11	Middle of left femur	Buck's extension	Died in 3 weeks from delirium tremens.
213	B. McV.	57 years	F	9–26–07	Lower ⅓ right femur	Buck's extension	Died in 1 week from broncho-pneumonia.
214	J. F.	55 years	M	11–9–13	Upper ⅓ right femur	Buck's extension	Died in 3 weeks from pneumonia.
215	P. C.	59 years	F	8–15–13	Middle of left femur	Buck's extension	Died in 3 weeks from pneumonia.
216	J. H.	48 years	M	7–12–10	Middle of left femur	Buck's extension	Died in 3 weeks from delirium tremens.
217	M. M.	65 years	F	2–20–13	Middle of left femur	Buck's extension	Died from shock.
218	P. R.	68 years	M	12–28–09	Middle of right femur	Buck's extension	Died in 4 weeks from pneumonia.
219	W. W.	68 years	F	2–9–11	Upper ⅓ left femur	Buck's extension	Died in 6 weeks from pneumonia.
220	B. A.	65 years	M	3–11–07	Middle of right femur	Buck's extension	Died in 8 weeks from pneumonia.
221	M. B.	68 years	F	2–5–07	Left femur, subtrochanteric	Buck's extension	Died in 1 month from broncho-pneumonia.
222	C. S.	60 years	M	1–26–09	Middle of right femur	Buck's extension	Died in 2 months from lobar pneumonia.
223	A. P.	80 years	M	7–12–10	Middle both femoral shafts	Buck's extension	Died in 12 hours from shock.
224	H. W.	82 years	F	5–2–06	Middle of left femur	In bed. Sand bags	Died in 4 hours from shock.
225	A. R.	65 years	M	11–19–13	Middle	Buck's extension	Died in 5 days from pulmonary œdema and nephritis.
226	J. V.	80 years	M	1–27–12	Supracondylar	Buck's extension	Died in 5 weeks from Bright's disease.
227	A. McA.	78 years	M	7–9–08	Middle of right femur	Buck's extension	Died in 8 weeks from uræmia and œdema of lungs.
228	M. S.	84 years	F	11–11–10	Subtrochanteric	Buck's extension	Died in 5 days from acute cardiac dilatation.
229	N. R.	79 years	F	6–8–13	Bilateral fracture of both femoral shafts (middle)	Buck's extension	Died in 1 month from aortic regurgitation.
230	F. S.	80 years	M	5–9–09	Upper ⅓	Buck's extension	Died in 2 weeks from gangrene.
231	W. R.	98 years	M	7–3–13	Subtrochanteric	Buck's extension	Died in 1 week from senility.
232	H. J.	80 years	M	10–6–13	Middle	Buck's extension	Died in 1 month from senility.
233	A. J.	75 years	M	4–8–09	Upper ⅓ left femur	Buck's extension	Died in 4 weeks from pneumonia.
234	M. L.	82 years	M	6–2–05	Middle of left femoral shaft	Buck's extension	Died in 6 days from pneumonia.
235	W. H.	79 years	M	7–17–06	Supracondylar, right femur	Buck's extension	Died in 5 weeks from pneumonia.
236	M. N.	71 years	F	4–3–12	Lower ⅓	Buck's extension	Died in 1 month from broncho-pneumonia.
237	J. R.	70 years	M	2–5–14	Middle	Buck's extension	Died in 1 week from senile pneumonia.
238	A. G.	70 years	F	4–10–11	Middle	Buck's extension	Died in 1 week from hypostatic pneumonia.
239	A. W.	72 years	F	2–9–13	Upper ⅓ left femur	Buck's extension	Died in 1 week from hypostatic pneumonia.
240	M.	80 years	F	9–5–09	Upper ⅓ right femur	Buck's extension	Died in 1 week from broncho-pneumonia.
241	H. O'N.	70 years	M	12–1–08	Middle of right femur	Buck's extension	Died in 1 month from pneumonia.
242	S. W.	80 years	M	5–3–10	Middle of left femur	Buck's extension	Died in 4 weeks from pneumonia.

leading hospitals of Philadelphia this fact has been recognized, and during the past six months for the word "cured" on their discharge blank has been substituted a report of the exact condition as regards the deformity and function of the affected limb.

In the hope of determining with some degree of accuracy the ultimate prognosis of simple fracture of the femoral shaft I have examined the records of these cases of seven Philadelphia hospitals: the University, Methodist, Episcopal, Jewish, Jefferson, Pennsylvania, and German, for the years 1904 to 1914. This examination comprised all cases treated in the wards of those institutions for simple fracture of the femoral shaft.

The list of cases thus collected numbered 400 and an effort was then made to look up each patient at his home. Of these it was possible to obtain the end-results in 242 patients, either by visiting them in their homes, or because death occurred while the patient was in the hospital. One hundred fifty-eight patients could not be traced.

The points investigated in each case were: (1) the site of the fracture, (2) the method of treatment, (3) duration of extension, (4) length of stay in bed, (5) period spent in chair, (6) duration of crutch and cane, (7) condition at the time of discharge, (8) the condition at the present time, (9) the effect on earning capacity.

Of the 242 cases tabulated below 96 were children under 16 years and 146 were adults.

TABLE I

Age	Number of Patients	Recoveries
Under 2 years	14	12
2 to 13 years	72	62
13 to 25 years	19	7
25 to 40 years	23	6
40 to 60 years	48	3
60 to 70 years	15	0
70 to 80 years	7	1

In the group of 14, 12 completely recovered. Of the two who did not, one (Case 13) had a paralyzed leg ever since the accident and at the present time (August 1914) 1½ inches of shortening, though at the time of discharge there was no shortening. In one (Case 14) there was considerable angulation and the child has to wear a brace. Ten of the fractures in this group were situated in the middle of the shaft, 3 in the lower third, and 1 in the upper third.

In the group of 72 between the ages of 2 to 13 years, 62 completely recovered. There were 3 cases of non-union in which open operations were performed (plated and wired). One (Case 176) completely recovered. The other two have shortening and one has some limitation of motion. In 7 cases there was an average shortening of one-fourth inch, one (Case 86) of angulation required braces. In this group 40 fractures were situated in the middle of the shaft, 19 in the lower third, and 13 in the upper third.

In the group of 19 cases between the ages of 13 and 25 years were 7 complete recoveries and 12 cases of shortening: in 1 case ⅛ inch; in 3 cases ¼ inch; in 2 cases ½ inch; in 4 cases 1 inch; in 1 case 1½ inches; in 1 case 2 inches. In this group 11 fractures were situated in the middle of the shaft, 6 in the lower third, and 2 in the upper third.

In the group of 23 cases between the ages of 25 and 40 years there were 6 complete recoveries, 16 of shortening, and 1 of non-union. The latter (Case 128) was treated by open operation and plating and at present has one inch shortening. The amount of shortening in the 16 cases is divided as follows: in 3 cases ¼ inch, in 7 cases ½ inch, in 1 case ¾ inch, in 2 cases 1 inch, in 1 case 1¼ inches, in 1 case 2 inches, and in 1 case 2½ inches. In this group 11 fractures occurred in the lower third, 9 in the middle of the shaft, and 3 in the upper third.

In the group of 48 cases between the ages of 40 and 60 years only 3 recovered completely, while in 45 there was shortening divided as follows: in 2 cases ¼ inch, in 4 cases ½ inch, in 6 cases ¾ inch, in 10 cases 1 inch, in 1 case 1¼ inches, in 12 cases 1½ inches, in 2 cases 1¾ inches, in 5 cases 2 inches, in 1 case 2¼ inches, in 1 case 2½ inches, and in 3 cases 3½ inches. In one (Case 157) measurements were not recorded because there had been shortening from a previous injury. In one (Case 159) there was non-union treated finally by open operation and plating. In two (Cases 135 and 165) a chronic ulcer remained on the tip of the heel of the fractured leg. In this

group 26 fractures occurred in the middle of the shaft, 10 in the upper third, and 12 in the lower third.

In the group of 15 cases between the ages of 60 and 70 years there were no complete recoveries. In 1 there was non-union, in 14 the shortening was divided as follows: in 1 case ½ inch, in 1 case ¾ inch, in 4 cases 1 inch, in 2 cases 1½ inches, in 2 cases 1¾ inches, in 2 cases 2 inches, in 1 case 2½ inches, in 1 case 2¾ inches. In 7 the location of the fracture was in the lower third, 4 in the middle of the shaft, and in 4 in the upper third.

In the group of 7 cases between the ages of 70 and 80 years only 1 (Case 192) completely recovered. There was shortening in 6 divided as follows: in three ¾ inch, in two ½ inch, and in one 1 inch. In 3 the location of fracture was in the middle, in 3 in the upper third, and in the lower third in 1.

The 44 deaths which occurred were as follows:

TABLE II

Age	Number of Deaths	Totals
1 to 5 years	1 shock, 1 acute gastro-enteritis, 1 tuberculosis	3
20 to 40 years	2 shock, 2 pneumonia, 1 delirium tremens	5
40 to 60 years	2 shock, 3 pneumonia, 5 delirium tremens	10
60 to 70 years	1 shock, 5 pneumonia	6
70 to 100 years	2 shock, 10 pneumonia, 2 senility, 1 gangrene, 1 aortic regurgitation, 3 general œdema, 1 nephritis	20

All cases were treated by extension, secured by weights attached to the leg and thigh by adhesive straps, the foot of the bed being raised to secure counter-extension. The weight in most cases ranged from 5 to 15 pounds. Sand bags were used to immobilize the parts. Children under one year were either treated by plaster casts or by extension with pulley attached the to framework over the bed.

CONCLUSIONS

The following conclusions seem tenable from the study of the foregoing cases:

1. In children fractures of the shaft of the femur involve mainly the middle third.

2. Ninety per cent of all cases of simple fracture of the shaft of the femur in children are followed by complete recovery.

3. In children at the time of discharge from the hospital (8 weeks) there is no stiffness in the knee.

4. The average period of treatment of children in bed and with crutches, splints and bandages is two to three months.

5. In adults fractures of the shaft of the femur involve mainly the middle third.

6. Between the ages of 15 years and 25 years there is about 50 per cent of complete recoveries, while after 25 years the permanent disability is proportionate to the age of the patient. Men above 40 years rarely regain strength and activity.

7. In adults in the absence of shortening there may be a lasting weakness of the leg and thigh. Permanent disability is as a rule associated with shortening, and is to an extent proportionate to the degree of shortening.

8. The average time in bed and with crutches, splints, and bandages in adults is eight months.

9. In adults at time of discharge (8 to 10 weeks) there is stiffness of the knee in 100 per cent of cases.

10. Among adult laboring men 90 per cent never become able to perform their regular occupations.

11. Among adult males not of the laboring class the great majority are able to perform their regular occupations.

12. The unsatisfactory results following the weight extension treatment of simple fracture of the femoral shaft in adults suggest that this method is either inadequate in itself or unskillfully applied.

13. Efforts for obtaining better results in adults should be directed to: (a) the employment of more weight in procuring extension, in the effort to entirely overcome the deformity; (b) early resort to open operation if extension seems inefficient; (c) the systematic use of massage and passive motion continued for months; (d) a longer use of crutches than is now customary; (e) education of the patient.

VAGINAL SUPRAVAGINAL HYSTERECTOMY FOR PROCIDENTIA AND LARGE CYSTORECTOCELE ASSOCIATED WITH CHRONIC FIBROSIS UTERI[1]

By HIRAM N. VINEBERG, M.D., F.A.C.S., New York

IT is fairly well established that vesico-vaginal interposition fulfills the object in view in most cases of procidentia and large cystorectocele when the climacteric is near at hand, or when the woman has already borne a good-sized family and further pregnancy may be eliminated. But the operation calls for a fairly normal uterus; in other words, if the uterus is very large through the existence of fibroid nodules or diffuse fibrosis, or is exceedingly small, through senile atrophy, it is contra-indicated. The same applies to those cases not so uncommon, in which the uterus, though of normal average size, is the subject of uncontrollable bleeding. The latter is usually observed in women approaching the menopause.

The method of procedure in chronic fibrosis associated with procidentia or cystorectocele has consisted either in a total hysterectomy, with the customary plastic upon the vaginal walls, or in the excision of a wedge-shape portion from the body and fundus. The first, that of total hysterectomy, possesses the objection that it leaves no solid tissue which can act as a support to the bladder. Whether this deficiency can be met by the technique of Goffe and Mayo, whereby the round ligaments and the broad ligaments are sutured together, I am unable to say from personal experience. This much may be said—that the technique in either case demands the greatest skill and the most extensive experience, and should, in any case, be limited, as C. H. Mayo[2] correctly states, to cases of the third and fourth degrees of prolapse with atrophy of the uterus and distention of the vaginal outlet. in which there is not enough solid tissue available for any type of an interposition operation.

The second method, that of excision of a wedge from the body and fundus, with which I have had considerable experience, fulfills the purpose in some cases and has the advantage in not creating the artificial menopause. But it has its drawbacks in that the uterus in many cases is too bulky and its walls are too thick and fibrous to lend itself to the procedure. Furthermore, in all instances it leaves a long line of suture in none too normal tissue, that does not always heal kindly and may be attended with profuse and prolonged suppuration. Still further, there may be very considerable oozing from the line of suture giving rise to a troublesome hæmatoma between the uterus and vaginal wall, if proper provision be not taken against it. In one case reported by Löwit, the bleeding was so great as to necessitate a subsequent hysterectomy.

A third vaginal method, which I have been practicing for over three years and to which I wish to draw attention, consists in amputating the body of the uterus fairly high up and utilizing the cervical stump as a *pelote* to hold up the bladder. When the woman is still comparatively young and the creation of the artificial menopause may be undesirable, the amputation may be done so high up as to leave together with the adnexa the lower segment of the uterus, with a corresponding area of endometrium. But not infrequently, a difficulty presents itself in that the vaginal portion is very much hypertrophied and elongated or badly lacerated with considerable eversion of the cervical mucosa. Where these conditions have presented themselves, I have amputated the lower portion of the cervix, leaving only a comparatively short cervical stump, extending from the vaginal vault to the lower uterine segment, and utilizing this for my purpose to support the bladder. After the redundant vaginal flaps are resected to the extent that the individual case demands, the cervical stump is sutured to the subpubic fascia and to the anterior vaginal wall. The bladder is

[2] Surg., Gynec. & Obst., 1915, xx, 253.

[1] Read before the Obstetric Section of the New York Academy of Medicine, February 23, 1915.

Fig. 1. (1) Left adnexa ligated and severed. (2) Ligature thrown around the uterine artery, knotted, but not tied.

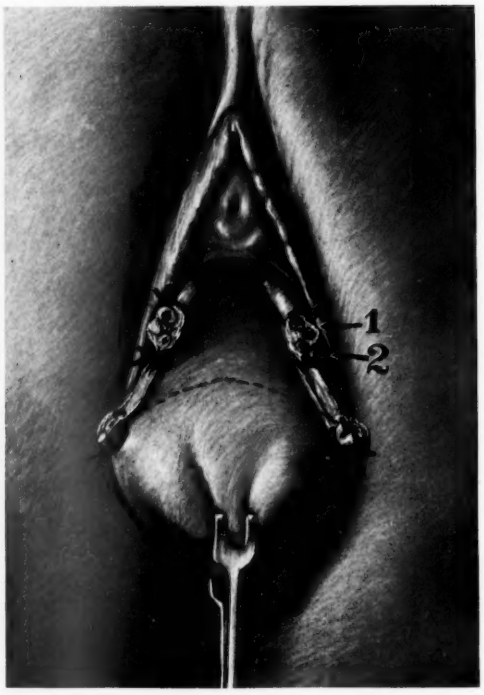

Fig. 2. Dotted line indicating the level of the amputation.

then forced to take a position within the abdomen and its prolapse is prevented by the firmly fixed cervical stump. The operation is completed by doing the necessary operation upon the posterior vaginal wall and perineum.

Löwit[1] as far as I can ascertain, was the first to employ the cervical stump after doing a vaginal amputation of the uterus as a *pelote* to hold up the bladder in cases of procidentia and cystocele. My technique differs from that of Löwit in the application of the subpubic suture (vide Fig. 3). This is passed underneath the mucosa and through the subpubic fascia, and has the effect of bringing the top of the cervical stump well up against the pubic arch. I deem this feature of great value. Another point of difference consists in excising most of the lower vaginal portion, which may be either very much hypertrophied and lacerated, or so elongated as to interfere with the results of the operation if left as it is.

The steps of the operation are as follows: (1) Longitudinal incision of the anterior vaginal wall, extending from near the urethral meatus to within an inch or an inch and a half of the cervical os. (2) Separation by sharp or blunt dissection of the vaginal walls from the underlying bladder. (3) The bladder is pushed up from the uterus and the base of the broad ligaments by gauze or scissor dissection. (4) Transverse incision of the vesico-uterine peritoneal fold. (5) Delivery of the body of the uterus through the vaginal incision. Up to this, the steps are identical with those followed in vesico-uterine interposition. (6) If the ovaries are to be preserved, a ligature is passed around the ovarian ligaments and the uterine end of the tube, on either side, and tissues cut

[1] Zentralbl. für Gynäk., 1911, xxxv, 297.

between the ligature and the uterus (Fig. 1). If the ovaries are to be removed, the ligature is passed around the infundibule pelvic ligament, containing the ovarian vessels. (7) A ligature is passed at the level of the os internum, to embrace the uterine artery on either side, Fig. 2. The body of the uterus is then amputated at the desired level by a wedge-shaped incision, the thin edges of the wedge pointing toward the cervix. The edges of the wound in the cervix are then brought together by a continuous or interrupted catgut suture, and the raw area of the cervix laterally is sutured in the same way. (8) Suturing of the cervical stump to the subpubic fascia and anterior vaginal wall as above described, Fig. 3.

Thus far I have had occasion to perform the operation ten times, the first operation being done over three years ago. Two of the operations were done during the past two months, and hence must be excluded for the present, regarding end-results. Of the other eight cases, one was under observation for three years and three months; two, for fifteen months; three, for twelve months; one, for eight months; and one was lost sight of after she left the hospital. The results in all the observed cases were excellent from an anatomic standpoint, and were good from a clinical standpoint, excepting in one. This patient continued complaining of an uncomfortable bearing-down feeling and from frequency of micturition, although there was no recurrence of the cystocele, but the vaginal portion of the cervix, which was very long, came down to the introitus. In this instance I should have done a partial amputation of the vaginal portion, as I had done in all of the other cases that presented long and hypertrophied cervices.

The value of the procedure was particularly well illustrated in the following case:

The patient, 23 years of age, had had an abdominal supravaginal hysterectomy done in an out-of-town hospital sixteen months before she was admitted to the Second Gynecologic Service of Mt. Sinai Hospital, on August 19, 1914. A couple of months afterward the patient noticed that everything came down, as before the operation. At the time of her admission, the long cervix with bladder and rectum were protruding from the vagina. August 21, operation. The anterior vaginal wall was incised from the urethral meatus to near the cervical os, the bladder was dissected away from the cervix and the base of the broad ligaments. On opening the peritoneal cavity a long fibrous band was found stretching from the cervical stump to the abdominal wall. This was severed. The redundant vaginal flaps were then excised, and after the cervical stump was shortened by a liberal excision of its lower part it was sutured to the subpubic fascia and to the vaginal wall, as described above. The operation was completed by a suitable posterior colporrhaphy. I examined the patient yesterday, seven months after the operation, and the result is simply perfect. The cervical stump is high up in the vaginal vault, both vaginal walls are in apposition, and when the patient bears down there is as little protrusion of the anterior vaginal wall as there would be in a normal nullipara. The patient felt perfectly well, and she stated that the parts felt as firm as before she had ever had a child.

Fig. 3. Cervical stump with subfascial suture (1) and vaginal sutures (2, 3, 4, and 5) in place.

MYOMA OF THE STOMACH

Report of a Case Successfully Removed

By J. E. JAMES, Jr., M.D., F.A.C.S., and S. W. SAPPINGTON, M.D., Philadelphia

THE warrant for a single case report is usually found in the rarity of the condition and this obtains in the present instance. While myoma is the third most important tumor of the stomach, it is so far behind cancer in point of incidence that, like sarcoma, the second in importance and frequency, it is a clinical and pathologic rarity.

More practical interest, however, attaches to another point. The relatively rapid increase in case reports of myomata of the stomach is an index of the vast increase in the number of abdominal operations. Without operative interference, gastric tumors and abdominal growths in general are often indiscriminately assumed to be malignant and hopeless. But further data in the matter of gastric myomata may outline a small but definite group of cases with perhaps suggestive symptomatology. These cases may be amenable to successful operative treatment, and such patients should not be allowed to dismiss operation as out of the question and progress unaided to a fatal end. Certainly the prognosis is not of the hopeless aspect of most carcinomata of the stomach.

Case. A freely movable, external leiomyoma of the stomach weighing 670 gr. and without metastases, removed by operation. Patient in good health and had increased in weight eight months after operation.

Clinical report. Miss B., age 63, white, school teacher. Claims to have always been in good health prior to present illness. In the past few years she has had "indigestion," the most prominent symptom being flatulency with epigastric distress. One year ago there occurred a severe gastric attack, the attending physician suspecting ulcer or malignancy. At this time there was epigastric pain, nausea, and vomiting without hæmatemesis and quite well-marked emaciation. After subsidence of the acute symptoms, there ensued a mild attack of diarrhœa with bloody stools. The patient, however, improved, gained in weight and appetite, and was able to resume school duties, though still complaining of more or less epigastric discomfort. In six months there was recurrence of the diarrhœa with blood in the stools, associated with abdominal distention. During this attack, a firm, movable tumor was discovered in the region of the umbilicus. Six months later, October 10, 1914, there was a further slight recurrence of the diarrhœa and abdominal examination by one of us (James) revealed a distinct, firm, rounded, non-sensitive mass, freely movable upon manipulation and change of posture. Pelvic examination failed to show any definite connection between the tumor and the pelvic organs. Unfortunately, no gastric analysis or blood examination was made.

The patient was operated upon October 19, 1914. Upon opening the abdomen the freely movable tumor was found attached by an apparently long pedicle to the anterior wall of the stomach near the lesser curvature and cardiac end. The gastric vessels were markedly enlarged and distended. The apparent pedicle was a tubular or pouched prolongation of the anterior wall of the stomach due to the dragging weight of the heavy tumor. The delicate capsule at the base of the tumor was split, the large associated vessels tied off, a small pedicle 1.5 cm. in diameter and similar in structure to the tumor ligated, and the mass removed. This left a small, protruding stump, difficult of differentiation from the stomach wall. This was removed, the 2 cm. area on the wall sutured and then inverted with a fine purse-string suture. The neighboring parts, the liver and glands especially, were carefully examined for evidence of metastatic growth, but none was found.

Eight months after the operation, the patient has been free of abdominal attacks, has gained considerably in weight, and states that she feels in first-class general health.

Pathologic report. The tumor was roughly the shape of a large potato somewhat flattened on two sides (Fig. 1). It weighed 670 gr. and measured 15x11x6 cm. It was enveloped by a very thin and delicate capsule. The color was grayish white with splotches of red. On section, numerous small hæmorrhagic areas were observed. Areas of fatty and mucoid degeneration were also in evidence near the central portion of the growth. Some of the larger hæmorrhagic foci exhibited central softening, but there was no definite cavity formation.

Microscopically, fields of interlaced bundles of long spindle cells with rod-shaped nuclei were fairly typical (Fig. 2). Areas of degeneration with hyaline change, dilated vessels and hæmorrhage were numerous (Fig. 3). No coagulation necrosis was observed and nuclear figures were not found. Connective tissue was relatively scanty. Dr. Thomas S. Cullen was kind enough to examine

several sections for us and expressed the opinion that it was probably a myoma and that the chances are about five to one that it was benign. He states that no absolute diagnosis can be made for the present and suggests that the condition of the patient after a few years should be watched with interest.

References to the literature of myomata of the stomach properly begin with Steiner (1), whose admirable monograph of 146 pages in 1898 is an exhaustive analysis of the subject. Steiner cites 23 cases. Further contributions are for the most part, as Hake (2) says, simply the reporting of additional cases. Hake himself, however, in an excellent review brought the matter up to date in 1912. Recent articles in American literature are by Thompson (3), Farr and Glenn (4), and Outland and Clendening (5). Farr and Glenn give an accurate list of reported cases and were able to increase Steiner and Hake's collection of 57 cases to a total of 84. The rapid increase of this list is indicated in the most recent summary of Nassetti (6) who has collected 140 leiomyomata of the stomach. We regret we were unable to see Nassetti's original article which is evidently a thorough treatise running through several numbers of *Tumori*. Deaver and Ashhurst (7) have a good textbook article with references.

Myoma of the stomach is in several ways unsatisfactory as a subject of accurate reference. Duplication or triplication of a single case occurs through confusion as to the proper assignment. It may be at one time ascribed to the operator, again to the reporter, and still occasionally again to a writer citing the case. Bland-Sutton's case, for instance, was really reported by Foulerton (8). The case operated on by Ehrenberg was reported by Perls and Neelsen (9) and cited by Cohn (10). Further, there is as yet no fixed standard under which the gastric myomata may be listed and the natural histologic features are in themselves confusing. Probably a number of myomata have been considered spindle-cell sarcomata, and vice versa sarcomata have erroneously been called myomata. Histologically, the structures of myomata in certain fields may suggest angiosarcoma, and Cullen noted in the case herewith reported that arrangement in some

Fig. 1. Photograph of tumor.

areas strongly suggested a perithelial angiosarcoma. Again, the occurrence of metastases might be thought to definitely class the tumor as malignant, but there is disagreement on this subject. The term malignant leiomyoma is hardly satisfactory, though embryologically the connection between sarcoma and myoma is naturally close. Another source of error in collecting myomata is due to reports covering myomata of more than one part of the gastro-intestinal canal or more than one variety of tumor, the caption of the article misleading one looking for myomata of the stomach only. Milovanovic (11), for example, in citing five cases of myoma of the œsophagus reports one extending into the stomach. Nichols (12) reports a case in which a carcinoma was also present. Yates (13) reports four sarcomata and one myoma under the title of "Sarcoma and Myoma of the Stomach."

In spite of the increased number of case reports we have as yet no suggestive symptom-complex as is apparently demonstrated by

Fig. 2. Microscopic field, high power, showing typical myomatous area.

Fig. 3. Microscopic field, low power, showing hæmorrhage and hyaline degeneration.

the fact, pointed out by Farr and Glenn, that a diagnosis is yet to be made before operation or autopsy. It may be, however, that the rarity of the occasion for such diagnosis is the cause of failure to keep the condition in mind. The clinical division of myomata of the stomach into internal and external varieties is eminently practical. The internal myoma in location and symptomatology is almost identical with carcinoma of the stomach and is nearly always mistaken for a malignant growth. Its long duration may be a point in differential diagnosis, but oftener the diagnosis is made at autopsy or after operation by the microscope. The external myoma should offer better prospects for diagnosis and treatment. Freely movable, large tumors with long pedicles, even allowing the mass to reach the pelvis, should be susceptible to the fine points of differential diagnosis and particularly to successful operation. It is noteworthy that while the tumor may actually be some distance from the stomach, the dragging on that organ through the pedicle makes the symptoms predominantly and significantly gastric and epigastric. The mass is variously diagnosed as tumor or cyst of the omentum, mesentery, kidney, or ovaries. The size is sometimes remarkable, as in the 6,000-gram growth reported by Perls and Neelsen. As might be suspected from the size and long pedicle of these tumors, nutritive conditions are commonly faulty, and degeneration, hæmorrhage, and cyst formation are frequent.

The case herewith reported presents nothing of peculiar interest. It may be said to be an average case. The object of the report is to remind diagnosticians and surgeons of the occasional occurrence of this growth.

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SPONTANEOUS RUPTURE OF THE SPLEEN[1]

By JOHN E. CANNADAY, M.D., F.A.C.S., Charleston, West Virginia

AN extended search of the literature in the Cincinnati Hospital Library, the John Crerar Library of Chicago, and the Library of the Surgeon-General's office in Washington, fails to disclose any reported cases of spontaneous rupture of the tubercular spleen, except one reported by Aufrecht (1). His case was a male 42 years of age, with an alcoholic and a tubercular history, who, while lying in bed in the hospital and talking to the orderly, complained of an unusual sensation in his left side. He requested the orderly to turn him over on that side, when he suddenly showed symptoms of severe shock and died in half an hour from the apparent onset. Post-mortem examination showed a transverse rent in the spleen 6 cm. in length. This rent involved almost the entire thickness of the spleen. From the description given, the spleen in this case undoubtedly was the primary seat of the tuberculosis.

Berger (2), in an extensive study of spleen injuries, collected all reported cases prior to 1902. He found 132 pathological, ruptured spleens, of which 93 were malarial; 15 only enlarged, no cause stated; 5 in typhoid; 1 in typhus; 1 in pneumonia; 3 in leukæmia; 1 in hereditary syphilis; 1 in liver cirrhosis from alcoholism; 9 in pregnancy; 1 in tuberculosis; 1 in hæmophilia; and 1 in varices of capsule of spleen. The tuberculosis case was Aufrecht's.

Senator and Litten (3) give as chief causes of rupture of the spleen, trauma and malaria. They mention among other causes typhus, typhoid, infarcts, pregnancy, hæmophilia, and anæmia, naming tuberculosis as a

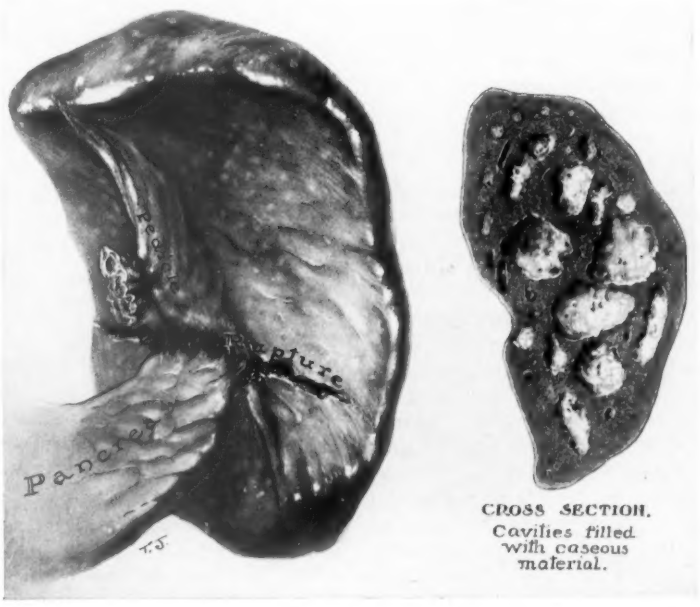

Fig. 1. Showing line of rupture of spleen. At right, section of spleen showing caseous nodules.

[1] Read before the Southern Surgical and Gynecological Association, Asheville, North Carolina, December, 1914.

possible cause, but cite only Aufrecht's case. Ciaccio (4) reports 9 cases from the literature and 1 of his own; no cases of rupture. Bayer (5) reviews cases from the literature as well as his own; no case of rupture reported. Laspeyres (6) cites 5 cases of splenectomy for tuberculosis, but no case of rupture. Villard and Santy (7) report 1 case of splenectomy for massive tuberculosis of the spleen. Fischer (8) reports 1 case of operation for tuberculosis of spleen with recovery; other cases cited from the literature; no rupture mentioned. Bland-Sutton (9) reports a splenectomy for a case of primary tuberculosis spleen. Stewart (10) reports a case of primary acute splenic tuberculosis, describes 1 of his own cases, and cites 33 from literature; no case of rupture. Winternitz (11) in his lengthy article on tuberculosis of the spleen, gives 1 case of his own, and a table of 51 cases from the literature, which he says includes all the cases published, except a few inaccessible ones. He does not give Aufrecht's. No cases of rupture. Death always follows if spleen is not removed. Recovery in 59 per cent of operated cases. Lorey (12) describes 1 case of primary tuberculosis of the spleen of his own, and reviews 32 from the literature, which he says are all he could find. He does not mention rupture in any of them. Strehl (13) reports 1 case of his own and reviews the literature. No case of rupture reported. Vorwerk (14) in an extensive essay on rupture of the spleen mentions only Aufrecht's case of the rupture of the tubercular spleen. Neither Rohrbach (15), Romann (16), nor Schrira (17) in their very extensive dissertations on this subject report any cases of spontaneous rupture of the tubercular spleen. Weil (18) in his review of tuberculosis of the spleen reports no cases. Von Bruns in his new *Handbuch der praktischen Chirurgie* says rupture of the spleen is very rare in tuberculosis, but does not cite any cases. Schubert and Geipel (19) describe one case of their own and cite 33 cases from the literature. No case of rupture. Garrow (20) says, "Primary tuberculosis of the spleen is rare. A few cases have been diagnosed prior to operation or autopsy. Secondary involvement is more common in children, less so in adults. In both forms the spleen is moderately enlarged; the parenchyma is soft, swollen, dark red in color, and scattered over with minute, round, gray tubercles. In the chronic form cheesy nodules, as large as hempseed or hazelnuts, may be rarely found, since death usually occurs before such changes have taken place. Splenectomy is indicated for tuberculosis of the spleen provided the diagnosis can be made and that the disease does not exist elsewhere, at least in a severe or active form." Johansson (21) in his very extensive contribution on the subject of "Spontaneous Rupture of the Spleen" was also able to find but 1 case of spontaneous rupture of the tubercular spleen, that reported by Aufrecht.

Primary tuberculosis of the spleen is uncommon, but occasional cases are seen. I found one post-mortem several years ago, and I wish to present a brief report of a case which I find to be an unusual one.

March 20, 1912, a patient, M. T. M., was brought to me by Dr. W. H. Wilson of St. Albans, West Virginia. He had been to Mt. Clemens for a month, taking treatment for what he supposed to be rheumatism and while returning to his home on the train, felt a sudden severe pain in the abdomen, which persisted. The patient became very weak and faint on the train, but managed to reach his home. Dr. Wilson was called and saw him four or five hours after the initial onset. He found him suffering pain, and shock morphia was given to relieve the pain. The patient had a most uncomfortable night and his pulse rate gradually increased. Dr. Wilson then brought the patient to the hospital, where I first saw him, and elicited the following history: The patient was a male 24 years old, barber by trade, had had the usual infectious diseases of childhood, was never robust, and always a little under weight. Had been feeling bad for more than a year but had continued to work at his trade most of the time. Had had occasional abdominal pains; urination had been rather frequent but he had not deemed his indisposition of sufficient importance to consult a physician until about six weeks before I saw him. At that time he was having occasional and irregular pains and tenderness in the principal joints of the long bones, along with some fever, and had diagnosed his case as one of rheumatism. He left his physician and on his own responsibility went to Mt. Clemens, where he remained for a month taking baths and other anti-rheumatic treatment. The joint pains subsided and he left for his home, a day's travel by train. He was lying on his back complaining of much pain on any motion, pulse 140, small volume, temperature 98° F., respiration 28 per minute, facies that of shock.

There was dullness in both flanks. A diagnosis was made of abdominal hæmorrhage and immediate exploratory laparotomy advised.

He was hastily prepared for operation and under ether anæsthesia, administered by Dr. B. S. Preston, a right rectus incision about 10 cm. in length was made. An abundance of free blood was present. The abdomen was explored and a search was made for the source of bleeding. On palpating the spleen, a decided rupture was felt on its median aspect. I compressed the pedicle between the thumb and the fingers of the left hand and made an incision about 17 cm. in length in the left side of the abdomen over the splenic area. The spleen was easily delivered, and its pedicle was clamped preparatory to removal. In bringing up the spleen it was noticed that the tip of the tail of the pancreas was adherent near the edge of the rent. The hæmorrhage had been very great and the abdomen was filled with free blood while near the spleen and extending for a short distance under its capsule was a considerable amount of dark, coagulated blood, evidently the result of a previous hæmorrhage, which took place under the capsule of the organ. The spleen was removed and its pedicle secured *en masse*, after which the principal vessels were each ligated separately, for further security. Aside from the spleen, the abdomen was found to be negative, with the exception of the right kidney, which was evidently completely disorganized from disease (probably tuberculosis), as it had the feel of a bag filled with fluid. Part of the free blood was sponged out and the two wounds were closed without drainage.

The spleen was about 15 cm. in length, 10 cm. in width and 3 cm. in thickness. There were numerous tubercles on its surface. In the body of the viscus there were a number of pockets containing caseous material. In some parts of the organ there were calcareous deposits also. The caseous masses varied from 1 cm. to 2.5 cm. in diameter. The tear in the spleen was distinct and ragged. It was fully 7 cm. in length and 1.5 cm. in depth at its deepest part.

The patient made a fair operative recovery. His wounds healed primarily. The pulse gradually subsided to 120. and the temperature, while fluctuating considerably, came down to nearly normal, and varied from 99° F. to 100° F. He left the hospital on the fourteenth day for his home. I urged on him the importance of having the right kidney removed, but he did not return to have this done. After arriving at his home, he gradually declined and died two months afterward. No post-mortem was obtained.

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THE APPENDIX

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AND
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THE general acceptance of the views of Barclay-Smith, Metchnikoff, and Sir Arbuthnot Lane, relative to the proximal colon and appendix, is becoming more and more apparent. By these men, this entire portion of the intestinal tract is regarded as useless, and a menace to man's well-being. They hold that, by reason of our modern regimen of dietary, colonic aid is no longer "a physiological necessity" to digestion, and is, therefore, a superfluous incumbrance, thus supporting the amendatory measures of Lane.

Keith (1) suggests, that "if the great bowel is as useless and injurious as is said, then medical men are face to face with a condition which threatens the health and survival of modern civilized races." He offers here an option against such radical opinions. "In place of appealing to surgery to adapt our digestive tract to our present dietary, it seems possible we may be able to discover a diet which is suited to our present digestive tract."

It is necessary, then, in compliance with the latter and more conservative view, to obtain so far as possible a correct knowledge of the nature and functions of this area. Unfortunately, as Keith remarks "we do not know, in any animal whatsoever, the exact functions of the great bowel; so far we have only guessed at them."

First, it is to be noted that the contents of the ileum undergoes a marked change upon entering the cæcum. Barclay-Smith (2) offered the first real tangible theory of the processes of digestion within this tract; also explanatory reasons for the existence of the so-called ileocæcal valve. His theory is based on the justifiable assumption that "all the digestive changes of food within the great bowel are due, not to any digestive secretion, but to the action of bacteria which find a permanent abode in the cæcum and colon." He made the inference, which has been justified by Hertz, Case, Carmen, George, Cole, and others that "the anastaltic movements which Cannon (3) had seen, fluoroscopically, to occur in the colon of the cat, also occurred in the upper portion of the human colon and that the purpose of the ileocæcal valve, so-called, was to prevent the contents of the cæcum and colon, which were undergoing bacterial digestion, from being forced by anastaltic movements back into the ileum."

It is quite generally believed that a limited power of digestion is retained by this portion of the large bowel, the proximal colon; that it has the physiological function of the absorption of fluids is also admitted.

Our knowledge of the mechanical factors of this area has been much enlarged by the researches of Cannon, Elliott, Keith, and Rutherford. In 1902, Cannon (3) first described the anastaltic movements already referred to; a series of delicate, or firm undulating contractions, beginning at a pulsating tonus ring, usually near the mid-transverse colon, and extending backward toward the cæcum, five or six per minute and normally each series of five to fifteen or twenty minutes duration. Thus the contents of the cæcum is retained for a period of time essential to completed digestion, and preparation for further disposal.

The chief factor in the induction of anastalsis is distention of the gut, and within limits, the greater the distention, the stronger the anastaltic impulse. Gas is ever present in the intestine, save in the newborn; especially is this true of the proximal colon, and it cannot be decompressed from this point in any direction, except into the appendix, for reasons explained later. "The presence of gas is a necessary correlative of the process of digestion going on in the cæcum."

Of special interest, are the observations of

Cannon (4), relative to the mechanical functions of the proximal colon. "The passage of material through the ileocæcal valve seems to stimulate the colon to activity. As a mass is nearing the valve, the large intestine is usually quiet and relaxed, though occasionally indefinite movements are to be observed; and sometimes, just before the mass reaches the end of the ileum the circular fibers of the colon, in the region of the valve, contract strongly so that a deep indentation may persist for several minutes disappearing as the muscles relax just previous to the entrance of the new material. The moment the semi-fluid content enters, strong contractions take place all along the cæcum and first of the ascending colon, pressing the material forward. These forces are met by deep anastaltic waves coming down from the transverse colon, sweeping back into the cæcum the advancing masses of food."

The subject of the proximal colon engaged the thought of John Hunter (5), who said: "If only we could understand the use of the ileocæcal valve, then we should understand the uses of the cæcum." As a result of a series of observations in 1903, Keith concluded that there was always a muscular sphincter at the junction of the small intestine with the great — "it is present in the amphibian bowel, the reptilian, avian and mammalian. When the great bowel undergoes retrogression as it does in bears, in their allies, and in many forms of insectivora and edentates, the ileocæcal valve is the only visible structure which remains to mark the junction of the small intestine with the large."

Keith's observations at this time concur with those of later authorities; Elliott, Barclay-Smith, Sir William Macewen, and A. H. Rutherford, the latter two deriving their opinions from studies on the living human subject through artificial openings of the cæcum. Rutherford's (6) observations were made through "an artificial anus in the cæcum 8 cm. long. This artificial anus was on the anterior wall of the cæcum, roughly 8 cm. from the head of the cæcum.

"*Cæcum.* The cæcum was collapsed, and its interior was occupied by pink folds of mucous membrane. The interior could be observed, also the ileocæcal orifice when it came into view. *Colon.* The interior of the colon appeared paler than the mucous membrane of the mouth, being more of a pink color. The interior was smooth, except where the mucous membrane lay in folds. There was no appearance of sacculation in the wall of the cæcum or colon. *Appendix.* At no time was the orifice of the appendix to be observed, on account of this case being an inflammatory one." *Ileocæcal valve.* As described briefly, we shall observe the ileocæcal orifice presenting a mammillary eminence of hemispherical contour, 1.8 cm. in diameter, scarlet in appearance, smooth and glistening, the summit truncated and pierced by an orifice of a stellate appearance with lobulated elevations between the rays of the stars. "This elevation is about 0.5 to 1 cm.

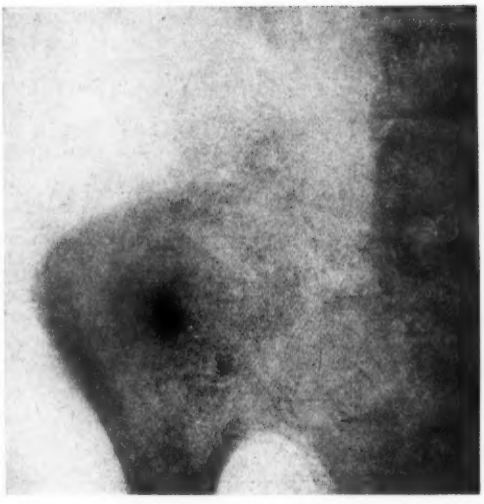

Fig. 1. On the third or fourth day following a bismuth meal, it is not unusual to find a small residue in the cæcum, the rest of the colon having been completely emptied. This residue is often about the size of a button and represents, I believe, stasis about the base of the appendix. This spot of bismuth is especially likely to appear after the appendix has been removed. In such instances, it seems to correspond to the portion of cæcal musculature involved in the purse-string invaginating suture. (As described by Dr. James T. Case,[1] New York Medical Journal, July 25, 1914.)

[1] Under date of June 3, 1915, Dr. Case writes: "I do not think that this little shadow is held about the base of the appendix because of any sphincter action, but merely because a certain amount of cæcal musculature has been put out of business by the purse-string suture." A view at variance with those of the author.

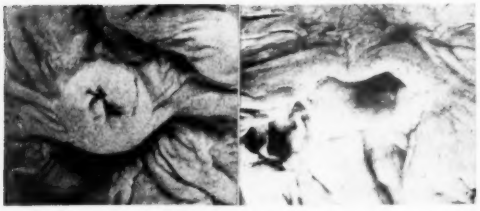

Fig. 2a (left). Male, dead 24 hours. Average temperature of room at time of death, 50°F. *b*, Male, dead 24 hours. Average temperature of room at time of death 60°F. Photographic plates from *The Ileo-Cæcal Valve*, by A. H. Rutherford, M.D.

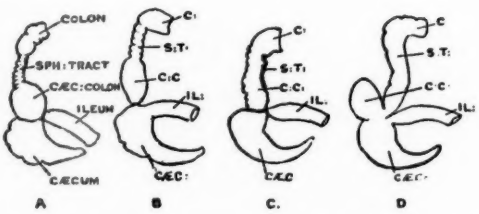

Fig. 3. "Figures illustrating four functional states of the ileocæcal region of the bowel of rats. In *A* and *C* the ileocæcal sphincter is closed; in *B* and *D* it is relaxed. In *A* and *B* the sphincteric tract is closed; in *C* and *D* it is open. Various conditions of the cæco-colic sphincter are shown in *A*, *B*, *C*, and *D*. (As described by Arthur Keith, *British Medical Journal*, December 7, 1912.) Corresponding activities were observed in the appendix in Case 2.

high." Surrounding the mammilla valve, or papillary eminence, of scarlet appearance, is the pink colored mucous membrane of the colon and cæcum, which lay in superimposed folds about the orifice. The valve mammilla was not to be observed at all times, only occasionally when peristalsis was more active within the ileum than usual, or when the interior of the cæcum was irrigated by warm water. "While peristalsis was moderate, the orifice of the papilla would open sluggishly, and the fæces would be discharged sluggishly; but, while peristalsis was active, the papilla would open quickly and the fæces would be ejected in jets, after which the papilla would close again smartly. At times flatus would escape, the papilla opening to allow its passage, or fluid mixed with flatus. The sphincter action was, however, perfect in control, and would allow nothing to pass without relaxation, not even flatus." This point is of greatest interest, for here, as suggested by Keith, Cannon and Elliott, Rutherford finds the function of the ileocæcal sphincter to be that of regulating the flow of a constant semi-fluid content from the small intestines and to prevent regurgitation of material back into the ileum. It was also noted by Rutherford that the exercise of this function seemed to be an automatic one, the same as that of the pyloric or internal anal sphincter, and without sensation, in function, but especially sensitive was the papilla to touch. The orifice was unyielding to attempts at dilatation, there being an elastic resistance, also similar to that of the internal anal sphincter. The papilla was not connected by any bands, ridges, etc., to any part of the interior of the cæcum, there was no appearance of frenula. *Pari passu* with the enlargement of the papilla, the orifice would open, apparently by relaxation of the circular muscular fibers, and semi-fluid fæces, about a drachm, would be observed to emerge from the opening with a force varying with the strength of the peristalsis. Usually the discharge of fæces was obscured by reason of folds of mucous membrane. Occasionally, during active peristalsis, the papilla would emerge "shifting its position by a series of rhythmical movements, of a peculiar sinuous nature, as might be those of a snake whose head was fixed in cotton sheeting."

Peristalsis. "On account of the aperture forming the artificial anus in the cæcum, peristalsis was probably less marked." Trauma and inflammatory conditions are well-known factors in the inhibition of these forces. Psychic factors may be mentioned also.

Chloroform. Of pathological interest are the factors of chloroform action, and the post-mortem changes which take place with specimens as ordinarily obtained. "Under deep anæsthesia, the firm, elastic mass altered, and the dimpled orifice relaxed. Instead of a mass there was now a slitlike orifice 2 ½ cm. long, through which the index and middle fingers were readily passed. The circular muscular fibers could now be felt as thickened bands at the margin of this orifice."

Fig. 4. Four and a quarter hours after ingestion of barium.

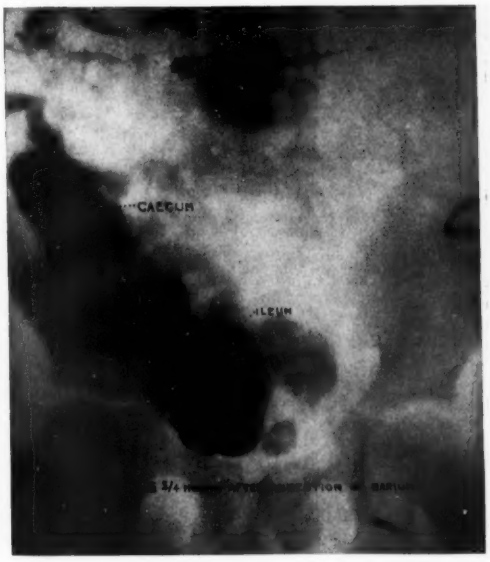

Fig. 5. Five and three-quarters hours after ingestion of barium.

It was found by Rutherford, as previously suggested by other writers, that, in order to preserve the continuity of specimen structures, it was necessary to maintain a certain low temperature of the room or surroundings in which death of the subject occurred — 50 to 60° F. The specimens were then removed immediately, and the arterial system injected with formalin. It has been shown that the circular muscular fibers of the intestine undergo rapid post-mortem changes.

Briefly we have come to see that there is an elaborate neuromuscular mechanism which regulates the passage of chyme into the cæcum.

"The significance of the ileocæcal orifice and sphincter is clear, if we admit they mark a point in the alimentary tract where one form of digestion ends and another begins." A form of rather complex peristalsis was observed by Rutherford, which he relates with diffidence. In the region of the cæcal wall, roughly 6 cm. from the papilla, four small dimples appeared on observation, the force causing them apparently originating in the muscular layer of the cæcum. These four dimples formed the corners of a rhomboidal figure if they were connected by lines. The first two to appear were about 4 cm. apart along the circumference of the bowel, the other two about 3 cm. nearer the head of the cæcum, and 4 cm. apart along the circumference of the bowel. Immediately these four appeared, there was a twitch, and the four corners approached as if by the contraction of a muscle connecting them, acting in the same manner as a purse-string. This immediately relaxed, and the depression of the corners disappeared . . . . as probably connected with or dependent upon this type of peristalsis, it should be mentioned that while observing the interior of the cæcum during that period that the valvular papilla was not visible, a pencil of firm fæces, 7.5 mm. in diameter and 6 cm. in length, was one day seen to be pressed out of an apparently round orifice in the wall of the cæcum. This was pressed out with considerable force in a direction at right angles to the surface of the bowel, evidently molded by the circular aperture through which it emerged. After the fæcal mass had been extruded, no orifice,

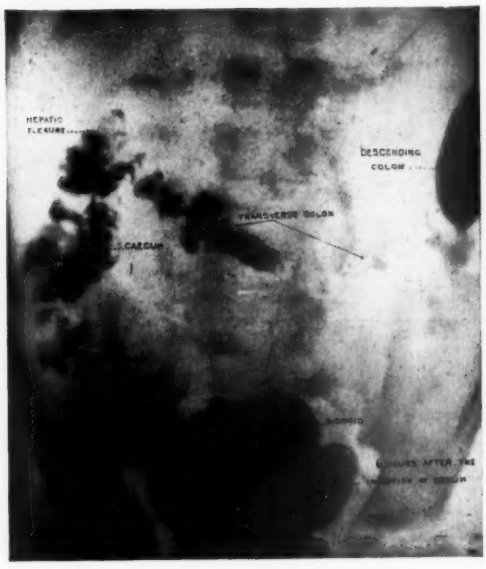

Fig. 6. Six hours after ingestion of barium.

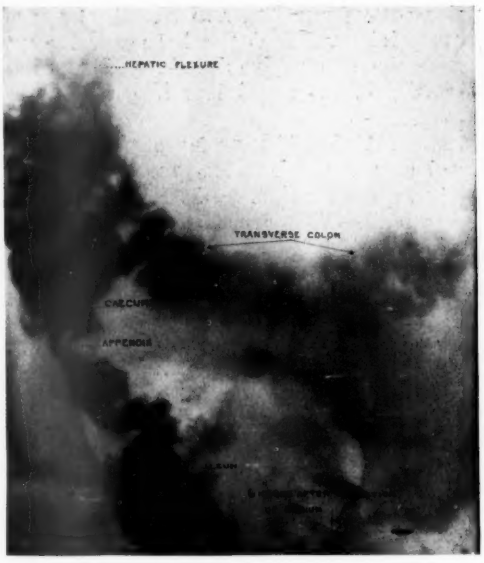

Fig. 7. Six hours after ingestion of barium.

however, could be discovered, and the spot from which it came had the same appearance as the rest of the intestine." The nurse in charge made frequent note of a similar occurrence. This observation suggested to Rutherford, that "below the caput cæcum coli a process of taking the semi-fluid contents discharged through the ileocæcal valve into pouches seems probable. In these pouches the semi-fluid contents are inspissated and then pressed out of these pouches into the lumen of the bowel, as pencil-like bodies, which being pressed together, form the characteristic lobulation observed in the fæces." As to the origin of these fæcal molds, I would venture the inference that, instead of being inspissated in pouches of the cæcum, which is highly improbable owing to an ever distended wall, these were molds of fæces inspissated within the lumen of the appendix and that in the instance as noted, "the extruding pencil-form molds" were the product of an impaired functionating appendix, its orifice being retracted beneath the superimposing mucous membrane of the cæcal wall, the same as that of the mammilla of the ileocæcal sphincter during quiesence.

Apart from the practical interest of these observations, an analogy of the functions of this and adjacent areas seem to the author apparent, thus affording a justifiable reason for an extended review. The provision for sphincteric control, the character of intestinal movements, the periodical discharge of ileal content into the cæcum, obviously are but manifestations which obtain throughout the entire intestinal tract, and suggest a similar office of appendix function.[1]

The musculature of the appendix, when not inflamed, is highly tonic and contractile in its nature; its neuromusculature as complete and perfect as that of other areas of the gut; its blood and nerve supply, which is the same as that of the small intestine, is abundant, especially so at the cæco-appendiceal juncture, at which point the lumen is much narrowed, as observed in young and healthy subjects. Ochsner and Percy have recently called attention to the latter observation. (*New Manual of Surgery*, p. 282.)

[1] Certain well-defined locks apparently guard the progress of food through the various areas of the intestinal tract. These locks under perfect sphincteric control, exercise their function through some kind of neuromechanism not fully understood. It is the authors' contention that at the cæco-appendiceal juncture this same sphincteric mechanism is maintained and exercises an important function in the designed fac tors of appendix activities.

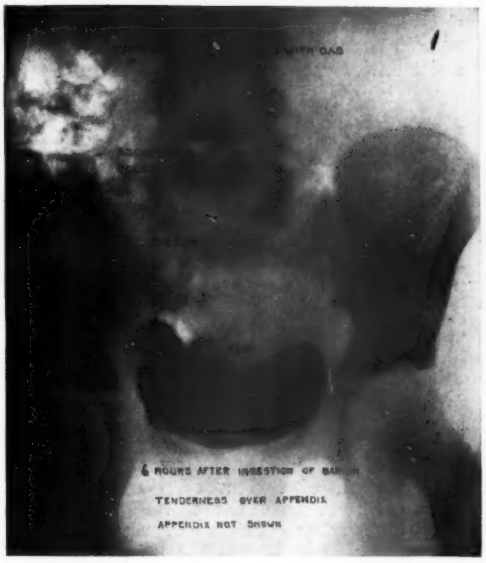

Fig. 8. Six hours after ingestion of barium; tenderness over appendix which is not shown.

Fig. 9. Five and three-quarters hours after ingestion of bismuth.

A clinical observation of November 23, 1913, (7), inspired our efforts, to establish, if possible, the functional nature of the appendix. It is to be recalled that, in the case referred to, the essential feature reported, was that of inflation which took place during the operation just as the cæcum and appendix were being delivered in the usual manner. The subject, a child of 11 years, not being deeply anæsthetized, began straining, thereby forcing gas from the distended cæcum into the appendix which, in this instance, became inflated to a point near bursting.

A tonic constriction at the base of the appendix proved competent to resist any manual pressure exerted; deflation was possible only when the anæsthesia was carried to a point beyond that of abolishing muscular reflexes, as was apparent by the relaxed recti muscles, when it suddenly gave way, and the member assumed the size and contour of the normal. No bands, membranes, or kinks were present; within the lumen, however, was found a No. 6 bird-shot, which according to the history given by the mother must have been an occupant of the appendix for a period of two years; no wild game nor any other known means of conveying the shot to the child's digestive tract could be recalled by the parent, except that two years ago, shot were used for the purpose of cleaning beer bottles and then thrown away. During this time, at least, the child suffered three or four quite severe attacks of gastric distress, the one previous to the operation being intense. While straining at stool the child was seized with cramps, became faint, and was carried to the bed, her condition as I found her bordering on collapse. Ochsner and Kelly (8), have for many years, recognized gas-distention of the appendix. In this instance, as with other though rare conditions, it may be presumed, that the appendix had become a retention cyst by reason of the presence of the foreign body, this being retained by sphincteric control at the base of the appendix. The sphincter during the previous faint or in the primary anæsthesia had relaxed and the cyst was emptied of its content and thus became an inflatable sack. Or by reason of the pressure exerted by exaggerated anastalsis and sphincteric forces within the cæcum, induced by the

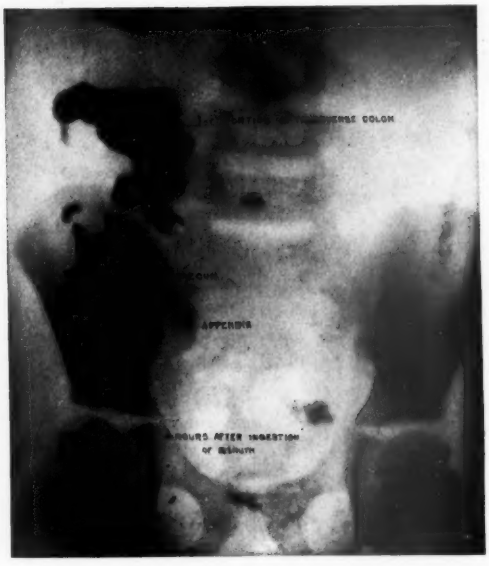

Fig. 10. Six hours after ingestion of bismuth.

shot or irritative influence reflexly, the appendix may have become distensile, gradually losing its normal "tone." Be that as it may, however, the point of greatest significance was the sphincteric control,[1] as described.

Cannon's work verified by Case, Carmen, and others in the human, together with Keith's and Rutherford's observations, form a basic foundation of physiological principles which need no further comment. Clinical observations of the past two years, case histories, and the examination of a large number of young children suggested, during these efforts, that to obtain definite evidence of the appendix musculature, it would be necessary to employ röntgenological methods of observation. Also, that the normal and healthy appendix might be observed, it was quite imperative that our subjects be children, very young persons, or persons absolutely free from any abnormality of the appendix whatsoever.

This procedure became possible through the efforts of Dr. L. G. Cole, who, for this purpose, suggested a "field-day clinic," to consist entirely of such subjects, as might be supposed normal and free from abdominal disease.

Through the courtesy of Dr. E. D. Brown, New York, twenty-seven children were furnished, being loaned by their several parents for the day, March 6, 1915. This clinic took place in the Laboratory of Dr. Cole, under whom the work was carried out, and to whom we are indebted for the following report which as appended invites a most critical study.

Case 1. Age 16½ years. *Röntgenologic findings.* Cap shown distinctly. Two and one-fourth hour röntgenograms show the stomach half empty; active peristalsis; cap well developed; sphincter normal; dilatation of the duodenum—possibly caused by pressure. Head of barium column in terminal portion of ileum. Six hour röntgenograms show stomach incompletely evacuated, with the head of the column of barium in the transverse colon, which is prolapsed. There is no evidence of an appendix.

Fluoroscopic findings. Head of the barium column in the ascending colon; large amount of barium in terminal coil of the ileum obscures the appendix. Appendix not shown.

Physical examination. Appendix, positive tenderness; right kidney prolapsed; nocturnal enuresis; skin, muddy; slight acne.

Case 7. Age 9 years. *Röntgenologic findings.* Barium in transverse colon in the six hour röntgenograms. Appendix distinctly shown crossing the ileum one-half inch from cæcum. Ileal stasis.

Fluoroscopic findings. Barium in the transverse colon and ascending colon. There is a dilatation of the terminal portion of the ileum. The appendix crosses the ileum about one-half inch from the ileocæcal valve, and then passes over toward the median line. There is an area of constriction at the point where the appendix crosses over, but it is doubtful whether this is organic or spasmodic.

Physical examination. Appendix, negative.

Case 10. Age 12 years. *Röntgenologic findings.* Cap shown distinctly. Two and one-half hour röntgenograms show the barium in the terminal ileum, and the stomach incompletely evacuated Six hour röntgenograms show the stomach completely evacuated. The appendix is not shown.

Fluoroscopic findings. The cæcum is incompletely filled. A dilated coil of ileum lies to the left of the cæcum, and the appendix shows distinctly, passing downward and to the right in a semicircular manner. The appendix itself is extremely tender and "sore like a boil."

Physical examination. Appendix, positive, active inflammation.

[1] Clinically confirmed by Dr. A. J. Ochsner, recently, in an appendix where "the entire mucous lining was gangrenous, and in which a long fæcal concretion was firmly grasped by this valve. By means of gentle manipulation the fæcal concretion was forced back into the lumen of the appendix, and immediately a gush of gas and fluid emptied into the cæcum."

CASE 26. Age 16 years. *Röntgenologic findings.* Cap shown distinctly. Two and one-half hour röntgenograms show the head of the barium column in the terminal portion of the ileum, with a slight trace of the chyme in the stomach. Six hour röntgenograms show the stomach empty, with the head of the barium column in the first portion of the transverse colon, which is prolapsed. Appendix shown distinctly.

Fluoroscopic findings. The cæcum is well filled. The appendix is incompletely filled, except when the barium is pushed into it from pressure on the cæcum. After the release of the pressure, the appendix vacates itself so that it is difficult to observe it.

Physical examination. Appendix, negative.

CASE 24. Age 9 years. *Röntgenologic findings.* Cap shown distinctly. Two and one-fourth hour röntgenograms show the barium in the ileum. Six hour röntgenograms show the head of the barium column in the transverse colon, and the cæcum well filled. The base of the appendix is shown distinctly.

Fluoroscopic findings. The cæcum, ascending and transverse colon are filled with the barium, and there is a small trace of the chyme in the ileum. The appendix shows distinctly; is hook-shaped, with the hook pointing toward the right and remaining constant. Pressure on the cæcum failed to distend the lumen of the appendix, The hook of the appendix is retrocæcal and shows very distinctly when the cæcum is pulled upward and to the left.

Physical examination. None made.

CASE 27. Age 11 years. *Röntgenologic findings.* Well-defined cap and pyloric sphincter. There is a spasm of the pyloric end. Two and three-fourths hour röntgenograms show a fleck in the region of the appendix. Six hour röntgenograms show the head of the barium column in the transverse colon, and the tail of the column in the cæcum. The appendix is shown indistinctly to the left of the cæcum.

Fluoroscopic findings. There is a small fleck of barium apparently about one-half inch below the cæcum. This has the appearance of a fleck in the appendix, although upon manipulating the cæcum, which is freely movable, none of the barium can be forced into the appendix. However, one can definitely say that this fleck is in the appendix. The entrance of the ileum into the cæcum is distinctly shown, and apparently is normal.

Physical examination. Appendix, negative.

CASE 9. Age 7 years. *Röntgenologic findings.* Cap not shown. Two and one-half hour röntgenograms show the head of the column in the terminal portion of the ileum. Six hour röntgenograms show the head of the barium column at the splenic flexure There is a dilated coil in the terminal portion of the ileum. The base of the appendix is shown, and possibly the tip, to the left of the dilated ileum.

Fluoroscopic findings. The cæcum and ascending colon are well filled. Without any manipulation one is able to detect the appendix passing out to the left of the cæcum. There is a kink in the center of the appendix, which is shown very distinctly upon manipulation.

Physical examination. Slight tenderness in right quadrant.

CASE 22. Age 11 years. *Röntgenologic findings.* Cap shown distinctly. Two and three-fourths hours röntgenograms show the head of the column in the terminal portion of the ileum, and the stomach empty. Six hour röntgenograms show the head of the barium column in the sigmoid, and the tail of the column in the cæcum. The appendix is shown distinctly.

Fluoroscopic findings. The head of the barium column is in the sigmoid. The ileum is completely evacuated, and the ascending and transverse colon are incompletely filled. There is a segmented area in the region of the appendix which is constant, but the dilated portions between the segments are larger than are shown in an ordinary appendix. The local point of tenderness is directly over this segmented area. There is some question as to whether or not this is the appendix.

Physical examination. Appendix, slight tenderness.

CASE 20. Age 12 years. *Röntgenologic findings.* Cap shown distinctly. Two and three-fourths hour röntgenograms show the head of the column in the ileum. Six hour röntgenograms show the head of the column in the transverse colon, and the tail of the column in the terminal portion of the ileum. The appendix is not shown.

Fluoroscopic findings. The cæcum and ascending colon are well filled. There is a moderate dilatation of the terminal portion of the ileum. The entrance of the ileum into the cæcum is well shown. There is no evidence of an appendix. However, pressure and manipulation on the cæcum expels the contents into a retrocæcal appendix, which is entirely behind the cæcum.

Physical examination. Appendix, marked tenderness.

CASE 16. Age 7 years. *Röntgenologic findings.* Pyloric end of the stomach incompletely filled. In the two and three-fourths hour röntgenograms the head of the barium column is in the terminal portion of the ileum. Six hour röntgenograms show a dilatation of the terminal ileum, with the head of the column in the ascending colon. There is no evidence of an appendix—probably obscured by the dilated ileum.

Fluoroscopic findings. The cæcum is well filled and there is a considerable dilatation of the terminal portion of the ileum—greater than in any of the preceding cases. The entrance of the ileum into the cæcum is well shown. There is no evidence of an appendix. Upon manipulation, the expulsion of the barium from the cæcum shows a retrocæcal appendix, passing out to the right across the ileum about one-half inch from where it joins the cæcum.

Physical examination. Appendix, slight tenderness.

CASE 11. Age 8 years. *Röntgenologic findings.* Cap shown distinctly. Two and three-fourths

hour röntgenograms show a retention of the barium solution in the cap. At this time the head of the column is in the terminal portion of the ileum. Six hour röntgenograms show the head of the column in the ascending colon. The appendix is shown distinctly, and is dilated and angulated.

Fluoroscopic findings. The cæcum and ascending colon show distinctly. The appendix is observed upon manipulation, and is well filled.

Physical examination. Appendix, negative.

CASE 19. Age 9 years. *Röntgenologic findings.* Cap and duodenum shown distinctly. Stomach shown to be empty in the two and three-fourths hour röntgenograms, and the barium is accumulated in the ileum. Five and three-fourths hour röntgenograms show the head of the column in the ascending colon, and there is a dilatation of the terminal portion of the ileum. The patient moved in these last röntgenograms. The appendix is not shown.

Fluoroscopic findings. The ileum is empty; the cæcum is well filled; the appendix is observed upon manipulation, but it shows much more distinctly when the barium is expelled from the cæcum. It has a cork-screw appearance—passing either upward or downward—probably the latter.

Physical examination. Appendix, very slight tenderness.

CASE 21. Age 7 years. *Röntgenologic findings.* Cap shown fairly distinct, as is also the sphincter. Two and three-fourths hour röntgenograms show the head of the column at the hepatic flexure and the tail of the column in the jejunum. Six hour röntgenograms show the barium in the cæcum, ascending and first portion of the transverse colon. A kinked appendix lying to the right of the cæcum shows distinctly.

Fluoroscopic findings. The cæcum is incompletely filled. The appendix shows distinctly without manipulation, passing to the right and with a loop passing upward.

Physical examination. Appendix, very slight tenderness.

CASE 12. Age 7 years. *Röntgenologic findings.* There is no evidence of the cap. Two and three-fourths hour röntgenograms show the stomach incompletely evacuated, and the barium scattered through the jejunum and ileum. Five and three-fourths hour röntgenograms show the head of the barium column in the transverse colon, and the tail of the column in a dilated coil of ileum. The cæcum is well filled. The base of the appendix shows distinctly.

Fluoroscopic findings. Considerable ileal retention; cæcum shows distinctly. A fine line, which is fairly characteristic of an appendix is well shown, but manipulation fails to fill or distend it. That is probably the appendix. The patient complains of considerable pain in this region when pressure is exerted.

Physical examination. Appendix, tenderness.

CASE 5. Age 5½ years. Röntgenologic evidence of a kinked appendix.

Fluoroscopic findings. The cæcum and ascending colon are well filled. There is no evidence of an appendix.

Physical examination. Appendix, negative.

CASE 14. Age 8 years. *Röntgenologic findings.* No evidence of cap. Duodenum shown distinctly. Five and one-half hour röntgenograms show the head of the barium column in the cæcum. The appendix is not shown.

Fluoroscopic findings. Appendix, extremely doubtful.

Physical examination. Appendix, negative.

CASE 4. Age 10 years. *Röntgenologic findings.* Five and one-fourth hour röntgenograms show a collection of barium in the cæcum. The appendix is well shown.

Fluoroscopic findings. The cæcum is filled. There is some evidence of barium in the base of the appendix.

Physical examination. Appendix, negative.

CASE 15. Age about 7 years. *Röntgenologic findings.* Cap incompletely filled. Duodenum shown distinctly, especially in the one and one-half hour röntgenograms. Four and one-fourth hour röntgenograms show the barium in the terminal ileum. Barium not advanced enough for appendix to be shown.

Fluoroscopic findings. No evidence of appendix—probably obscured by ileum.

Physical examination. Appendix, slight tenderness.

CASE 2. Age 11 years. *Röntgenologic findings.* No evidence of appendix in any of the röntgenograms.

Fluoroscopic findings. The barium in the cæcum shows distinctly, as it does also in the appendix, which is of medium size. The appendix is alternately of greater and lesser density in various areas, and at certain periods of the examination the apex of the appendix is well filled. At times there appears to be motion in this organ, but I am not absolutely certain that this is independent of the peristalsis of the adjacent viscera. After manipulation of the cæcum (at which time the appendix is well filled) the tendency to an evacuation of the appendix is evident.

Physical examination. Appendix, slight tenderness upon deep pressure.

CASE 3. Age 12 years. *Röntgenologic findings.* Stomach and first half of appendix shown distinctly in the röntgenograms.

Fluoroscopic findings. No fluoroscopic examination. *Physical examination.* Appendix (?)

CASE 13. Age 12 years. *Röntgenologic findings.* Stomach incompletely filled. Two and one-fourth hour röntgenograms show the barium scattered through the terminal portion of the ileum, which is dilated. Five and one-fourth hour röntgenograms show that none of the barium has entered the cæcum, and therefore the column has not advanced enough for the appendix to be shown.

No fluoroscopic examination.

Physical examination. Appendix, marked tenderness.

The relative frequency with which involvement of the appendix and adjacent structures is encountered in young children is at once apparent from a thoughtful analysis of the foregoing report. Clinically, our case reports when obtainable refer in most instances to such an early-life history, in whole or part as follows: Cross peevish and colicky babies; later gastric and gastro-intestinal disturbances, facial pallor, especially about the nose and mouth — halo oris; œsophageal, gastric, and duodenal spasm — "belly and side-aches, bilious attacks, fever spells," nausea, sometimes vomiting, in fact all the typical evidences by the laity, attributable to "worms"; nervous manifestations, frequently convulsions; unhealthy general appearance; acne, erythema, and other cutaneous suggestions of "auto-intoxication," all of which are of clinical significance, regardless of local pain or tenderness. Obviously, of greatest importance is the early recognition of the initial lesion, which, we feel justified in believing to be essentially that of early life, developmentally or inflammatory.

In determining these and allied conditions, the fluoroscope affords our only certain avenue of intelligence, for by methods devised and employed in modern röntgenoscopy, we believe a more accurate knowledge of conditions within the abdomen may be acquired than by even exploratory incision, incidentally, supporting a conservational regard against possible or needless risk.

Our efforts have thus far dealt mainly with functional activities of the appendix, to establish which we have reviewed the subject of the mechanical factors of the adjacent structures, and in our clinical, röntgenological, and fluoroscopical reports, we have given, briefly, evidences which suggest that the same principles obtain within the appendix musculature as elsewhere along the intestinal tract, and therefore command the same recognition.

Our observations confirm the inferences of Keith, and as suggested by Prof. Berry; viz., that the appendix is a specialized part of the cæcum, and we believe its relation, functionally, with the structures of the ileocæcal region of no less importance. We have demonstrated within its musculature a definite sphincteric and peristaltic function as observed both clinically and fluoroscopically (Case 2.) It is likewise apparent that, in the normal appendix, fæcal material is retained during quiesence of the cæcum and adjacent viscera; i.e., from one period of digestion to another, and thus, inferentially, that these retained "fæcal molds" (not long-retained, dried-out, or inspissated), may, or do become, incidentally or purposefully, a culture medium providing automatically fresh bacteria for colonic digestion; in brief, that the appendix is a normal physiological "culture tube."

If as we have endeavored to show, the appendix is possessed of the specialized functions as described, are we not including a potential factor to post-operative ills, in the employment of the usual method of its removal? We are supported by Dr. Ochsner in the belief that not the slightest portion of the sphincteric structures should be invaginated, a careful resection being the method of choice.

I desire in conclusion to acknowledge the service of Dr. Edward Archibald, whose insistence and counsel inspired these efforts. Especially would I express my appreciation of the interest and collaboration of Drs. Cole and Ochsner, by whose indulgence, corroborative evidence has been made possible.

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APOPLEXY

Suggestions for More Rational Methods of Dealing with Spontaneous Intracerebral Hæmorrhage Based upon the Pathological and Physiological Features[1]

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SPEAKING generally, apoplexy may be considered as an acute pathological process associated with a more or less general intracranial physiological disturbance, rapid or gradual in its progress, which *may terminate fatally*, and with disturbances of function due more directly to the local effects of the lesion such as hemiplegia, hemianopsia, and the like, *possibly entirely potential at first.*

The arteries to the lenticular nucleus and external capsule are small branches arising chiefly from the middle cerebral a short distance from its origin with some assistance from the anterior and posterior cerebral. One of the larger of them runs along the outer side of the lenticular nucleus, where it is covered with the external capsule—a disposition which may have something to do with the occurrence of the larger hæmorrhages so likely to take place just outside this nucleus and into the substance of the hemispheres. The arteries of the optic thalamus arise from the posterior communicating or the posterior cerebral. The larger hæmorrhages spring from these sources (1).

The incidence of rupture follows closely the order of arterial diseases, yet favors so strongly the arteries of the central ganglia as to occur in the lenticulo-striate and lenticulo-optic arteries in 75 per cent (Durand-Fardel) of such cases (2).

Brissaud (3) graphically shows the arrangement of these important arterial branches and their relation to the outlying structures.

Edes (4) has compared the locations of intracerebral hæmorrhages in their order of relative frequency, giving the Durand-Fardel and Boston City Hospitals records together (see Table I).

The size of such cavities may vary from pea-size to that of a whole hemisphere. The larger hæmorrhages may be found in the region of the central ganglia (Oppenheim), and centrum semi-ovale while the cortex, pons, and cerebellum usually harbor the smaller lesions.

The lesion is nearly always solitary when due to diseased arterial walls (Charcot), and the intra-arterial pressure, transmitted directly to the cerebral tissue, forms an hæmorrhagic cavity, either by *destructive laceration*, when we may expect the contents of the cavity to be composed of blood and lacerated cerebral tissue, by *simple pressure without destruction*, the contents then being pure blood, or by a combination of both such processes (5).

TABLE I

	Durand-Fardel	Boston City Hospital.
Corpus striatum and optic thalamus, together	22	4
Corpus striatum	13	10
Optic thalamus	5	3
Corpus striatum, optic thalamus, and middle lobe	2	1
Corpus striatum, optic thalamus, and a considerable portion, not well defined, of the hemisphere	12	2
Corpus striatum, optic thalamus, with posterior lobe	1	
Corpus striatum and middle lobe	3	3
Corpus striatum with a considerable portion of hemisphere	5	1
Corpus striatum with an extended portion of base of ventricle	1	
Optic thalamus with middle lobe	2	1
Optic thalamus with posterior lobe	1	
Middle (parietal) lobe	19	1
Sphenoidal horn of middle lobe (temporal)	2	4
Anterior (frontal) lobe	11	3
Posterior (occipital) lobe	11	5
A not well-determined extent of one hemisphere	4	3
The superficies of the convolutions (once with corpus callosum)	3	1
Insula		2
Cortex (not further defined)		1
Small multiple		3
Meninges (secondarily)	31	6
Ventricles	66	8
In the cerebellum right lobe	6	
In the cerebellum left lobe	5	4
In the cerebellum middle lobe	2	
In the protuberance (pons)	13	4
Protuberance, crus cerebri, crus cerebelli, external capsule, fourth ventricle		1

[1] Read before the Atlanta Neurological Society, September 9, 1915.

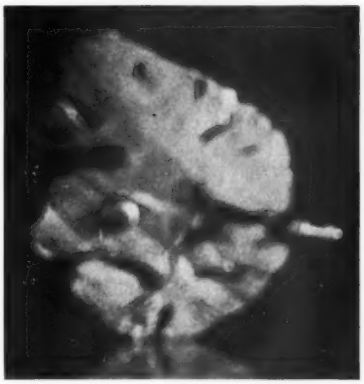

Fig. 1. Cross section of right brain at genu, showing relation of insula to basal ganglia and capsule. Needle passes through corpus striatum into capsule.

Farther reaching than the boundaries of the cavity itself are the effects of the hæmorrhage. The walls of the hæmorrhagic cavity, whether pressed aside or lacerated show hæmorrhagic infiltration which may be quite marked enough to simulate, in the immediate vicinity, smaller separate hæmorrhages (6). Peripheral to the walls a reactive inflammation occurs, and serous infiltration and œdema may be quite marked. Microscopically, the tissue here shows not only these processes, but also, frequently, damaged and lacerated nerve-elements. *Further away, the nerve-tissue may be not actually destroyed, but may be subjected to pressure which temporarily, at least, may cause physiological inhibition which further, may lead to permanent pathological destruction, should the pressure persist.*

The destruction or interruption of the nervous conduction paths or the ganglia themselves due either to laceration or *to pressure* will lead eventually to *secondary trophic* degeneration which in turn terminates by supplementing sclerosis for normal tissue along the tracts distal to the destruction lesion.

When the hæmorrhage passes beyond certain close confines, the general intracerebral pressure may be great enough to force the cortex firmly against the inner cranial surface and obliterate the convolutions and sulci, or similarly, the falx may bulge toward the opposite side. A cavity in the state of formation may burrow in various directions, possibly breaking through the ventricle, or toward the cortex or into the crus cerebri.

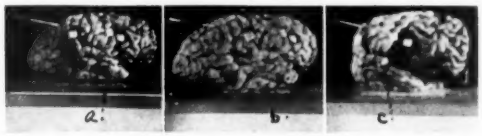

Fig. 2. Reconstructed brain showing needle passing into lateral cerebral fissure about junction of this fissure and projected Rolandic fissure. *a*, Lateral view; *b*, posterior view; *c*, anterior view. (Dotted line leads to outer end of trochar.)

The original seat of the hæmorrhage, then, may be in the striate body or in the optic thalamus, or in the internal capsule. It may burst through the ganglionic boundaries, and involve the capsule secondarily in its destructive lesion with a resultant hemiplegia. The cortex may be involved in the process. The excavation may proceed to the ventricle and break through its walls, causing diplegia or some of the serious results of intraventricular hæmorrhage; the optic radiation may be destroyed so should life persist hemianopsia will result. The nuclei of the cranial nerves may be disturbed or destroyed and secondary degeneration occur, should the hæmorrhage course toward the crura and pons, while hæmorrhages of greater size, which may involve the larger part of the whole hemisphere, may permanently destroy or throw out of gear the whole cerebral mechanism.

Apart from the symptoms of actual destruction we must consider interference with physiological functions due to pressure, inflammatory reaction, œdema, anæmia, or infiltration which may project similar symptoms, and from which complete recovery is possible should the inciting cause be removed. We must conceive that functional disturbances of neighboring structures, disturbances due to nutritional disorders, anæmia, and mechanical causes which may become pathological and permanent if the causative factors persist, may be interrupted in their existence by the removal of such elements and the existing conditions improve, while the secondary sclerotic changes may be obviated or partially obviated.

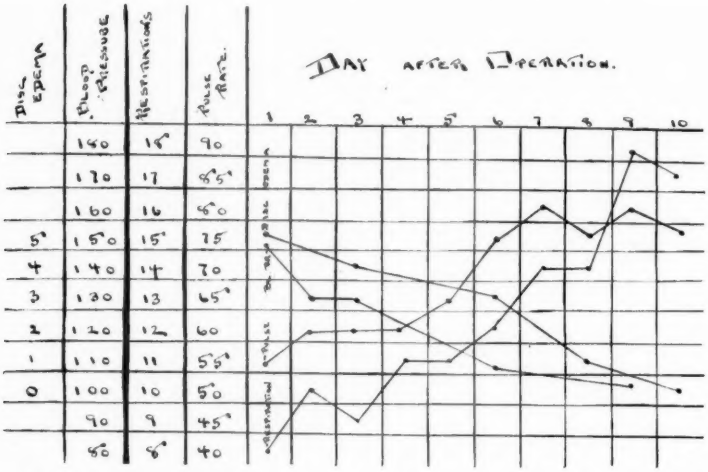

Fig. 3. Chart showing changes in pulse rate and blood pressure, respiration and disc œdema starting at a time immediately before operation and running to the tenth day after operation.

Cases in which hemiplegia exists under such circumstances are not out of the range of possibility — where the potential paralysis may be avoided entirely — cases which clear up more or less rapidly, either partially or almost completely, indicating the presence of neighborhood pressure with physiological inhibition rather than destructive lesions of the capsule itself. We may argue that in the cerebral tissue, irritation, inflammation, œdema, pressure, and infiltration may secondarily lead to sclerosis and destruction far reaching in their effects. May not the removal of the clot, the presence of which may cause the reactive œdema and pressure, do away far more surely with the disturbing reaction, and its serious secondary results?

Persistence of hæmorrhage in the cerebral tissue may mean a continuation of the cerebral excavation, and this in turn means that the dangers of a large hæmorrhage involving ventricles, nuclei, optic tracts, cortex, etc., threaten. In such an event an opening or safety valve may entirely obviate these sequelæ.

Destruction, then, may not be benefited directly by operative means, but potential destruction, at least, may be warded off by (1) relieving pressure, œdema, infiltration, or reactive inflammation, which if persistent may and usually will transform physiological disturbances into pathological destruction; and (2) by forming an artificial opening to the end that excavation may be stopped, and pontine, cortical, ventricular, and paths other than the drainage sinus may no longer be the *via minoris resistentiae*.

Perhaps more important in the consideration of spontaneous intracerebral hæmorrhage are the disturbances of intracranial pressure. With the arterial pressure, no longer confined by the vessel-walls, transmitted to the cerebral tissue direct and thence to the whole intracranial contents, we are confronted with a condition somewhat analogous to that seen in the case of a growing brain tumor. As the intracranial space is encroached upon, the pressure is conveyed to the various portions of the cerebrum, and the brain substance suffers from pressure anæmia. Reactive œdema may occur adding its part to the already increasing intracranial pressure.

At this period we may expect disturbances of function in the cardiac, respiratory, and vascular centers. Under such conditions also we may expect and do find the usual symptoms and signs met with in other con-

ditions which cause this intracranial pressure. Œdema of the discs (an extremely important diagnostic aid), a decreasing pulse rate, and slowed, perhaps stertorous, respiration together with a rising blood pressure, portray rather vividly the intracranial picture. The pressure anæmia of these vital centers, leading to protective reaction in the general system, raises the general blood pressure and forces blood more successfully through the compressed cerebral arteries, thus counteracting temporarily, at least, the anæmia of the centers. Finally, unless the existing disturbances subside, death will ensue unless the balance of pressure lies with the general blood system and remains high enough to tide over the abnormal conditions. Once the intracranial pressure has become so great as not to allow enough blood, even under the greatly increased general pressure, to be conveyed to the centers, the result is fatal.

To recapitulate, then, we are dealing with spontaneous intracerebral hæmorrhage associated with: (1) a condition of general increased intracranial pressure causing anæmia of the centers which may lead to death immediately or in the course of days; and (2) local destructive action of the blood under pressure with its immediate local effects and its delayed indirect effects. To combat these conditions, indications point toward (1) removal of the general pressure on the cerebral vessels, and (2) removal of the clot.

The muscle-splitting operation with vertical division of the temporal fascia, separation of the fibers of the underlying muscle and exposure of the underlying bone, according to the method first devised by Cushing, are the first steps involved.

With the trephine and rongeur an opening is made in the skull at a point corresponding to a surface projection of the basal ganglia (7) and insula, and the dura exposed. Inspection of this latter may reveal subdural hæmorrhage, should the blood have excavated out through the cortex, and absence of brain pulsation. A cross incision is next made in the dura and the flaps reflected. Cortical inspection may be of great importance. The presence of cerebral œdema, a tendency for the brain to herniate through the opening, an

Fig. 4. *a*, Left central facial paralysis showing as patient attempts to smile, three days after operation; *b*, seven days after operation. Note marked improvement.

increase in cerebrospinal fluid, and the signs of hæmorrhage, either cortical or subcortical, should be ascertained at this step. Whether the hæmorrhage is approaching the cortex or not is to be determined, as the method of operative approach may depend entirely upon this. Localized œdema or a discoloration or swelling or differences in consistency may indicate that hæmorrhagic excavation is taking place in this or that portion of the exposed cortex. With this hint, an exploratory puncture may be made with a trochar and blunt obturator. Should this reveal the presence of an hæmorrhagic cavity this may be dealt with by guiding a small rubber drain in along the tract of the trochar after slowly evacuating the clotted and semi-clotted blood from the cavity, if this be possible. The drain is brought out through the skin opening and removed, as indications warrant, during the early convalescence, probably in the first few days. Procedures are more complex where the cortical inspection does not point to the location of the hæmorrhage. Under such circumstances, it has seemed that exploratory cerebrotomy in the region of the corpus striatum and other basal ganglia (see preceding table) should be the procedure of choice. Sections of the brain, it will be noted (Fig. 1) indicate that the shortest path to the striate body is by way of the island of Reil. Theoretically, at least, this would appear to me the most promising avenue of approach. This leads to a consideration (1) of the physical difficulties in approaching the surface of the insula and, (2) of the

physiological characteristics of the intervening tissue.

Reconstruction of our brain sections indicate that the needle should pass almost directly in at about the junction of the projected sulcus centralis with the lateral cerebral fissure (Fig. 2). This involves careful separation of the edges of the operculum and guiding the needle into the depths of the lateral fissure taking extreme care that the middle cerebral artery in its course upward is not injured. This step will be the more easy as the separation of the temporal convolution (superior) from the upper opercular borders is the greater.

According to Dana, in the thalamic symptoms proper, central pain, anæsthesia, and hemianopsia are to be expected. The two latter may occur from extrathalamic lesions. Observations suggest vasomotor, temperature, and disturbances of the visceral centers in extensive lesions of the thalamus. The pain is usually in the arms and hands, next in the leg and foot, and rarely in the head, may be intense and is always on the paralyzed side. It is more a burning discomfort than a sharp aching pain. Dana has not noticed the paralyses of emotional expressions in any of his strictly thalamic cases.

Tendency of investigations indicate that the thalamus is essentially an organ for the reception and distribution to the cortex of sensory centers for vision, hearing, smell, equilibrium, and deep and superficial sensibility.

Sachs concludes the thalamic syndrome is characterized by (1) a slight persistent anæsthesia; (2) a slight hemiplegia usually without contractures and rapidly egressive; (3) a slight hemiataxia and a more or less complete astereognosis; (4) sharp pains on the hemiplegic side often persistent, paroxysomal, and intolerable; and (5) by choreic and athetoid movements on the paralyzed side.

Further views on the basal ganglia are numerous. Southard (8), Rhein (9), Rhein and Potts (10) and others have collected interesting data and much literature on the function of these bodies; tremors, sensory disturbances, and hyperkinesis being among the symptoms attributable to their disturbances. Others believe that the proximity of the internal capsule is, at least, partly responsible for some of the symptoms ascribed to the ganglia.

The basal ganglia, and the island, the claustrum, and the external capsule, the other structures involved in this approach, can hardly be considered as obstacles when compared from a functional standpoint with the internal capsule.

Having encountered the insular cortex the needle, with the trochar in place, is carefully inserted a short distance, the obturator removed, and if no hæmorrhage is encountered the needle is pushed deeper. In this manner, hæmorrhage is sought for both in the island, in the claustrum and external capsule before the striate body itself is explored. At a depth of between 1 and 2 cm., the end of the trochar will encounter the corpus striatum. Here, one must be careful that no clotted blood nor portions of brain matter clog the opening and screen the lesion. Should the findings be negative we may either guide the needle a fraction of a centimeter deeper into the internal capsule and thence into the ganglia bordering the inner side of this structure or we may attempt another puncture after removing the needle completely.

Exploration of the lateral ventricles, best following the lateral approach of Keen (11) which may be utilized in this field, may be an extremely important step especially in the presence of marked hyperpyrexia, and if the hæmorrhage has not been located elsewhere, as the frequency with which the hæmorrhage may burst through the basal ganglia into the ventricles — and the presence of intraventricular blood may be of very serious import — is somewhat underestimated.

In dealing with hæmorrhagic accumulations one must be careful to use drainage material, if this be necessary, which will cause the least reaction, such as rubber or gutta-percha.

In a case lately operated upon by us we were able to locate, upon cortical inspection, the hæmorrhage which had broken through the insular cortex and appeared between the boundaries of the lateral cerebral fissure. In this case the cavity was about 4 cm. in diameter, and extended deep into the ganglionic

area. Hence, in this case ganglionic puncture was unnecessary. Drainage together with the relief of the intracranial pressure by forming a decompressive opening in the manner aforementioned changed this condition of almost certain death to an almost normal state in the course of a few days, as will be seen by the following data.

This patient came to us six days after the onset in a comatose state with left hemiplegia. The classic signs of increased intracranial pressure, and the steadily progressing features of the case were important operative indications. Following the operation the disc œdema dropped from 5D. to normal; blood pressure dropped from 156 to 110; respirations rose from 8 to 18, and the pulse rate increased from 56 to normal (Fig. 3). The facial paralysis cleared up quite markedly (Fig. 4), and the leg paralysis cleared moderately, while the arm showed improvement. This case, however, had suffered from pressure for six days before relief, and it was doubtful in our mind whether the local destruction of the motor paths could be bettered at this late stage, yet the rapid clearing up indicated that the removal of the clot had had a beneficial effect. Two weeks after operation the patient was in excellent condition, and walking about with some difficulty. As it might be expected in case of such standing, better results can be expected in the relief of the general pressure than in the ganglionic exploration together with drainage, since the length of time elapsing between the onset and operation was great enough for the at least partial metamorphosis of physiological disturbances into destructive lesions.

Procedures such as have been suggested should be rapid and accurate. Careful handling of the cerebral tissue, delicate manipulation in inserting the drainage material, the application of the proper methods to avoid shock, careful technique, the administration of urotropin, and care in the after-treatment are presupposed.

Under the present technique, it is hardly to be considered that the dangers of operative procedures in such cases would overbalance any good that might ensue. It is hardly to be denied that the decompressive features of this operation as a life-saving measure are advisable (12). Whether the ganglionic exploration will be successful in alleviating the paralyses, as appears possible, can only be determined when this procedure has been applied to a larger series of cases, and at *earlier stages* in the course of the affliction.

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CHORDOMA, WITH THE REPORT OF A MALIGNANT CASE FROM THE SACROCOCCYGEAL REGION

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A CHORDOMA is a rare tumor. In searching the literature I am able to find reports of but fifteen cases. Therefore it is of interest because of its rarity. It is of still more interest because of its origin from notochord (chorda dorsalis) structure. Until recently it was regarded as a pathological curiosity. Recent cases, including the one which we shall report, indicate, however, that chordomata are also of clinical interest and importance.

Clinical history. A male, age 26, white, an American, was referred to the University Hospital at Iowa City, by Dr. F. H. Kisor, Kalona, Iowa, May 19, 1913. The entrance complaint was — pain in the rectum and difficulty of defæcation.

Family history, negative.

Past history. Patient has had measles and chickenpox. He denies having had syphilis or any previous bowel trouble. He is an abstainer of alcoholic liquors and uses tobacco moderately. In other respects the past history is also negative as regards the condition in question.

Present illness. On February 22, 1913, he was in a railroad wreck during which he sustained a fracture of the collar bone and received bruises on the head, right leg about the calf, and about the coccyx.

He had a dull aching pain in the coccygeal region ever since the injury. For some time the pain was not increased by bowel movements. About May 1, i.e., about 9 weeks after the injury, the pain became worse, and was increased at the time of bowel movement. At the same time he began to have difficulty of defæcation and the fæces were blood-stained. The symptoms became rapidly worse and on May 19, three months after the injury, he came on the advice of his physician, to the University Hospital for operative relief. On examination, a tumor mass, protruding forward from the posterior wall of the lower part of the rectum, was found. It seemed to occlude the lumen of the rectum to about two-thirds of its extent. It did not appear to be adherent to the coccyx nor to any other portion of the vertebral column.

The tumor was excised by Dr. William Jepson, the excision being made by way of an incision which extended from the anus to the tip of the coccyx. This relieved the obstruction. The patient improved rapidly and, for a while, did not require medication to secure movements of the bowels.

The patient left the hospital eight weeks after the operation, feeling that he had made a fairly good recovery although he was never, neither before nor since leaving the hospital, entirely free from a slight rectal discharge of pus and blood; nor was he ever entirely free from pain which was increased during acts of defæcation.

Two months after leaving the hospital, the pain, discharge of pus and blood, and difficulty of defæcation, all became worse. On the advice of his physician, he returned to the hospital. On examination, which caused the patient considerable distress, a nodular and somewhat ulcerated mass extending around the rectum in an annular manner and causing considerable constriction of the lumen of the rectum, was found. The mass was not movable. The X-ray examination was negative as were also the Wassermann, blood and urine examinations. A portion of the mass was removed for microscopical examination. This indicated, as did the examination of the first specimen, that it was a chordoma. Dr. W. R. Whiteis then performed a radical operation — Tuttle's modification of the Kraske operation — and removed three inches of the tumor-involved portion of the rectum. The growth was again found to be non-adherent to any portion of the vertebral column. The operation relieved the symptoms of obstruction and the patient improved for a while. Before long, however, there was a recurrence of the tumor with a return of the symptoms. These became progressively worse. Obstruction finally necessitated the performing of a colostomy which was done on March 26, 1914, 11 months after the tumor was first noticed, by Dr. C. J. Rowan. The incision was made just to the left of the left border of the left rectus. The peritoneal cavity was opened. The tumor was found not to have extended upward along the rectum very far. From this time on, the bowel contents were evacuated through the artificial anus thus produced. The patient became more and more emaciated and otherwise rapidly failed in health. He left the University Hospital June 9, 1914 and died a month later (July 10, 1914). It will thus be noticed that death occurred 14 months after the tumor was first recognized. No autopsy was secured.

Gross pathology. The piece of tissue removed at the time of the first operation was an oval shaped mass which measured 10 x 6 x 5 cm. Its surface contour was rather ragged. The cut surface indicated that the mass removed was made up in largest part of intestinal wall tissue surrounding the tumor mass. The tumor proper was, apparently,

fairly well defined from the surrounding tissue and occupied principally the submucosa. At one place it was covered by normal mucous membrane; at another place the mucous membrane had disappeared, leaving an ulcer. The muscularis was partly invaded by the tumor and partly pushed backward by the growing mass.

The tumor mass proper was 5.5 cm. long, 4 cm. wide, and 2.5 cm. thick. The tumor was very soft in consistency and of a light color. It was made up, for the most part, of rather homogeneous material, although there were numerous small holes varying in size from those just visible to those which measured one millimeter in diameter. These contained a soft jelly-like substance.

Microscopic pathology. The microscopic picture is rather striking and quite characteristic. The most obvious and diagnostically important feature is the presence of large vacuolated cells which tend to break up, forming a foamy substance. The cells occur in groups or columns, which are separated from each other by an interstitial substance which is quite homogeneous. Here and there are distinct bands of fibrous connective tissue. In the muscularis, the muscle fibers are separated by the infiltrating tumor-cells.

The following description is based on the study of a section stained with hæmatoxylin and eosin. The tumor-cells proper are large epithelial-like cells with a small, deeply staining nucleus and a large amount of faintly staining vesicular cytoplasm. Most of the cells are in a degenerated, vesicular, or vacuolated state. The few cells that seem to be intact, are rather isolated and present a fairly distinct outline and a granular cytoplasm. Cells in all stages of disintegration are present. Those which are but slightly involved are larger than the intact cells and present one or a number of clear areas — vesicles. When there is but one vesicle, it may be of large size, causing the nucleus to be flattened and crowded to one side of the cell, giving to it the appearance of a fat-cell. More often the vesicles are multiple. For a while the cell outline seems to be retained. Later, with the increase in the size of the vesicles, the cell outline becomes broken up, until finally we find a number of nuclei lying in a foamy mass of protoplasm with no cell outlines distinguishable. The changes described also occur in normal notochord cells, as they become mature. The larger of these areas of broken down cells represent the small cysts recognizable with the naked eye. The intact cells measure from 6 to 10 and the vesicular disintegrated cells from 10 to 18 microns in diameter.

The interstitial substance between the groups and columns of cells is rather homogeneous, taking a diffuse red stain with eosin. This is the substance that gives to microscopic sections of the tumor its cartilaginous appearance, and which caused Virchow to regard the tumor as cartilaginous in nature. The amount of interstitial substance varies greatly in different parts of a section — in some places it is in excess of the cellular elements; in others, it is greatly overshadowed by the latter. The interstitial substance consists partly of mucin as we shall note later.

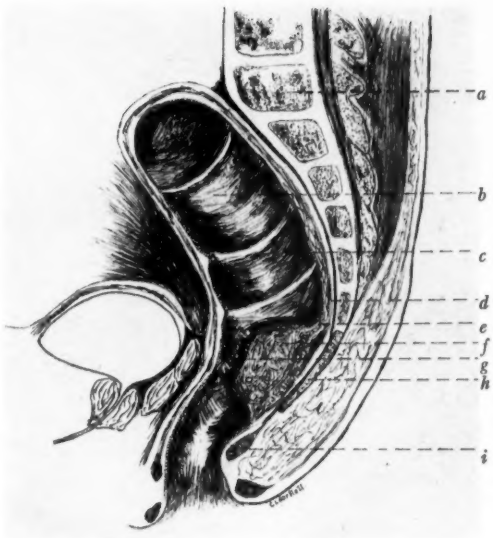

Plate 1, Fig. 1. Illustration representing the position, relative size, and general relationship of the chordoma to the surrounding tissue.

a, Sacrum; *b*, rectum; *c*, mucous membrane of the rectum; *d*, longitudinal muscular fibers of rectum; *e*, circular muscular fibers of rectum; *f*, chordoma; *g*, ulcerated surface of the chordoma; *h*, coccyx; *i*, internal sphincter muscle.

The fibrous connective tissue is arranged in bands between large areas of tumor substance. This tissue appears to be for the most part but incidentally present, representing the connective tissue which was present in the normal tissue before its invasion by the tumor. The same explanation applies of course, to the muscle fibers which are present.

Microscopically there is distinct evidence of malignancy as indicated by the infiltration of the adjacent muscle-tissue without any attempt at capsule formation.

Sections stained by Mallory's aniline-blue method reveals certain features not so well brought out in sections stained with hæmatoxylin and eosin. The vesicles of the large tumor-cells take a light bluish stain. The foamy substance formed from the breaking down of cells takes a somewhat more intense blue stain, as does also the more firm intercellular substance. At first thought, this was believed to represent collagen fibrils. It does not, however, occur in bundles of wavy fibrils such as the latter usually form. It appears to be principally mucoid in nature or at least in places contains

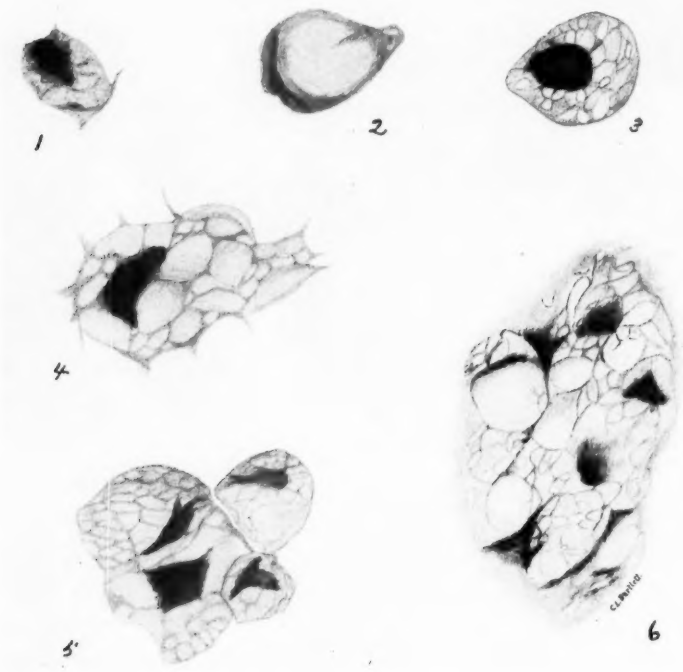

Fig. 2, (1) Intact chordoma cell. (2) Chordoma cell with a large vesicle in cytoplasm. (3) Chordoma cell with a number of small vesicles in cytoplasm. (4) Chordoma cell with indistinct cell outline due to vesicular disintegration of the cytoplasm. (5) A group of chordoma cells showing marked vesicular change of the cytoplasm. (6) A mass of chordoma tissue consisting of a number of indefinitely outlined nuclei in a mass of foamy substance which represents the vesicular disintegrated cytoplasm of a number of cells.

mucin, as indicated by its taking a reddish stain with thionin. The tumor contains no substance that reacts to fat stains.

CHORDOMATA: GENERAL DISCUSSION

It is now very certainly established that a chordoma is a tumor consisting of notochord (chorda dorsalis) tissue. This belief is based on the location of the tumor and on the similarity of histological structure. The notochord originates as a pouch from the entoderm, pushes upward and comes to lie just ventral to the neural tube. It represents the first axial skeleton. Around it is later formed the bodies of the vertebræ and the posterior portion of the base of the skull. The notochord extends from the lower part of the vertebral column up to the juncture of the occipital and sphenoid bones. It disappears from the bodies of the vertebræ during fœtal development but frequently remains as a small pulpy mass, the nucleus pulposus, in the intervertebral discs. Occasionally also, masses of notochordal tissue may be found anterior to the bodies of the vertebræ or as a small nodule in the clivus blumenbachi, projecting upward from the spheno-occipital synchondrosis.

Virchow (1), who first recognized the small masses of notochordal tissue in the clivus regarded them as tumor-like proliferations of vesicular degenerated cartilage-cells from the synchondrosis in question and gave to such the name — ecchondrosis physalifora, because of the vesicular cells surrounded by

Fig. 3. Photomicrograph (low power) representing the chordoma invading the submucosa and muscularis of the rectum. *a*, Mucosa; *b*, muscularis; *c*, chordoma.

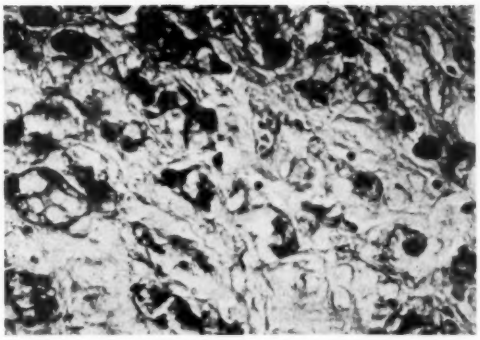

Fig. 4. Photomicrograph (high power) of chordoma showing intact cells, vesicular cells in all stages of disintegration, and intercellular substance.

a matrix of what appeared to be hyaline cartilage.

Mueller (2) was the first to express the view that these nodules had their origin from rests of notochordal tissue.

Ribbert (3) more conclusively proved that such was the case and gave to the process the name chordoma. He found that these small nodules, not larger than a pea, were of frequent occurrence, being found in about 2 per cent of all autopsies. These small nodules do not produce any clinical symptoms.

In the light of our present knowledge we do not regard these small masses as true neoplasms. They are rather to be explained as masses of notochordal tissue, possibly hyperplastic, that have been forced out of the bone during the process of fœtal development.

Grahl (4) reported the first case of chordoma which produced clinical symptoms. This tumor was located at the base of the skull in the clivus blumenbachi. It was a definitely circumscribed tumor, the cut surface of which measured 2 x 3.5 x 3.5 cm. and which was attached by a pedicle to the sella turcica. There was compression of the adjacent cranial nerves which had resulted in symptoms of paralysis.

Fischer (5) was the first to report a malignant chordoma. The term malignant is here used in the pathological sense in that the tumor was of an infiltrating character.

Klebs (6) was the first to report the occurrence of a tumor-like nodule of notochordal tissue away from the usual location at the base of the skull. In this case the mass grew anteriorly from one of the cervical vertebræ.

Mazzia (7) was the first to report a case of true chordoma growing elsewhere than at the base of the skull. The location was the sacral region.

To date, I have found reports of fifteen cases of what may properly be regarded as chordomata. The one which I report is the only one which has a history of traumatism that appears to be quite directly connected with the development of the tumor. If such a relationship exists it is not probable that the traumatism has in this case, caused any displacement of a portion of the notochordal tissue that remained in an intervertebral disc. It is much more probable that the trauma may have excited to growth, a rest of notochordal tissue that had been displaced into the soft tissue of the posterior wall of the rectum during the period of fœtal development. The lack of connection between the tumor and any part of the vertebral column would tend to favor that view.

SUMMARY AND CONCLUSIONS

1. Chordomata are tumors of interest because of their rarity and their origin from notochordal tissue; they are also of importance because of the clinical symptoms which they produce.

2. Nodules of notochordal tissue, not exceeding a pea in size, are found in about 2 per cent of all autopsies (Ribbert). These do not produce clinical symptoms and are not to be regarded as true neoplasms.

3. Including the one here reported, sixteen well recognized cases of chordomata have been reported to date.

4. Of the sixteen cases reported, ten had their origin from the base of the skull in the region of the spheno-occipital synchondrosis; six developed in the sacrococcygeal region.

5. Of the sixteen cases reported, ten were pathologically malignant and six, benign, although some of the latter caused death by virtue of their location. Of the malignant ones, six had their origin from the base of the skull and four, from the sacral region.

6. In two of the cases reported, the tumors appeared to be entirely independent of the vertebral column or base of the skull, suggesting their origin from rests of notochordal tissue displaced during the time of fœtal development.

7. Traumatism appeared to play a direct rôle in producing the tumor in one of the cases (Albert).

In conclusion, I desire to thank Drs. Kisor, Jepson, Whiteis, and Rowan for the clinical history of the case, and Drs. Royce, Grover, and Bartlett for assistance in the making of histological preparations, photomicrographs, and drawings.

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DEPARTMENT OF TECHNIQUE

A NEW OPERATION FOR UMBILICAL HERNIA

By F. G. DuBOSE, M.D., F.A.C.S., Selma, Alabama

A STUDY of the pathological anatomy of umbilical hernia and of the surgical principles at present adapted for its cure reveals an inefficiency of treatment as demonstrated by 15 per cent of recurrences. A review of the works, very early by Richet and recently by Moschcowitz, leaves but little to seek in its anatomical description. There appears to be a lack of appreciation of the rôle played by the tendinous and ligamentary supports of the umbilical fascia in the production of recurrences after operation. The linea alba at the junction of the third tendinous intersection of the recti is pierced by four apertures which normally close when the cord is tied at birth. The fibrous remains of the obliterated vessels entering these openings assume a new and different anatomical form and function; the umbilical vein becoming the round ligament of the liver, being incorporated in and forming a part of the suspensory ligament of the liver. The urachus and the umbilical arteries pass downward underneath the peritoneum following its reflection back of the symphysis pubis, joins, and acts as supports to the bladder, the urachus to the fundus and the arteries laterally. A dissection of these obliterated vessels demonstrates firm attachment to the fascia posterior to the pubes; it is convincing that from this fixed point to the umbilicus these three cords pull the umbilical fascia downward and backward. The round ligament of the liver exerts traction upward and backward on the umbilical fascia; its name has a tendency to obscure its dual function. The umbilical fascia is situated in the linea alba where the fusion of aponeuroses and fascia makes this area the most fibrous and least muscular portion of the abdominal wall occupying the center of a tendinous cross, one arm being formed by the linea alba the other by the transverse tendinous intersections of the recti.

The elements entering into the formation of the umbilicus or Richet's fascia are of structural interest; anatomically this fascia is reinforced in its entirety by interlacing transverse fibers (Richet). The openings for the exit of fœtal vessels are infundibuliform, so that when cicatricial closure has

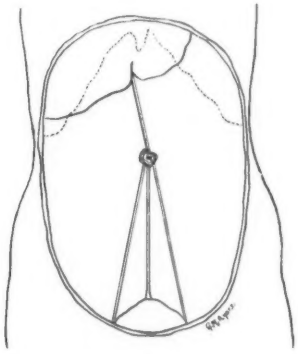

Fig. 1. Diagrammatic illustration of umbilical ligaments.

Fig. 2. Cross section showing lines of traction exerted by umbilical ligaments.

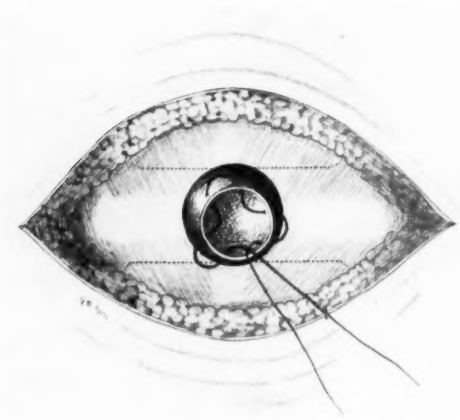

Fig. 3. Purse-string suture in stump of hernial sac, dotted lines indicating proposed transverse parallel incisions.

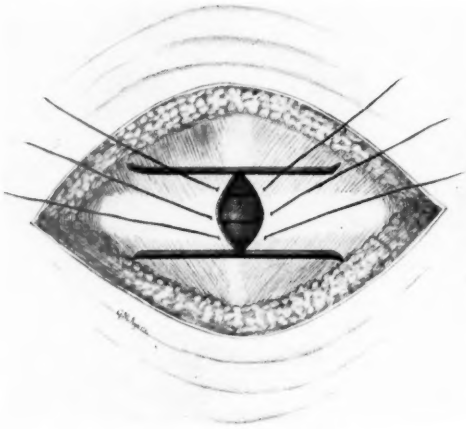

Fig. 4. Lateral flaps mobilized, sutures inserted.

occurred it is overlaced with fascial tissue in covering the openings (Moschcowitz).

There are six lines exerting traction on the umbilicus. It is a physical fact that the varying angles of traction thus exerted on this fascia make it normally an unyielding plane offering a maximum of static resistance to abdominal forces at this point. From without in the structures of the umbilicus are superficial cicatrix, fascia with four areas of imbedded cicatricial tissue, and peritoneum, thus differing from other parts of the abdominal wall in the absence of skin, subcutaneous fat, two layers of superficial fascia, and of preperitoneal fat.

The pathogenesis of the congenital type or true omphalocele is from arrest of development, the umbilical ring failing to close.

Moschcowitz demonstrates plausibly that the acquired form arises from a gap between the upper margin of the umbilical ring and the umbilical vein; the smaller amount of adventitia in the umbilical vein as compared with that of the umbilical arteries diverging below, and to the dense fusion of the obliterated ends of the arteries and vein filling the lower part of the um-

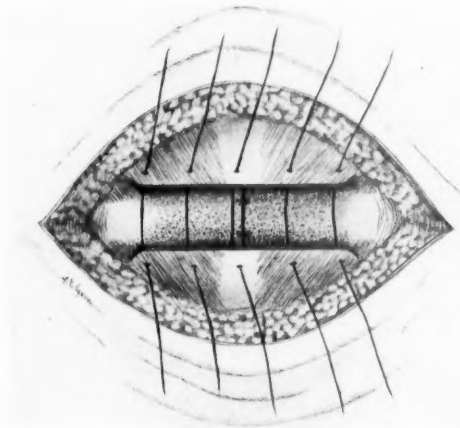

Fig. 5. Sutures inserted in upper and lower edges of aponeurosis.

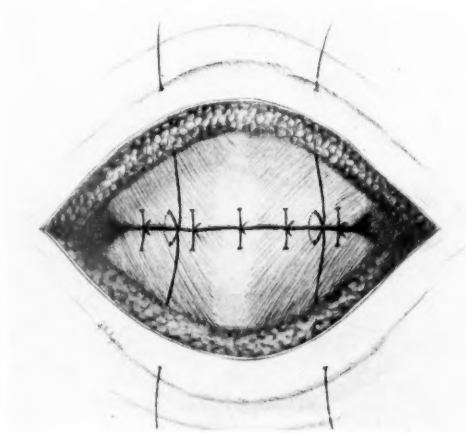

Fig. 6. Closure of superficial aponeurotic layer, with figure-of-eight sutures inserted.

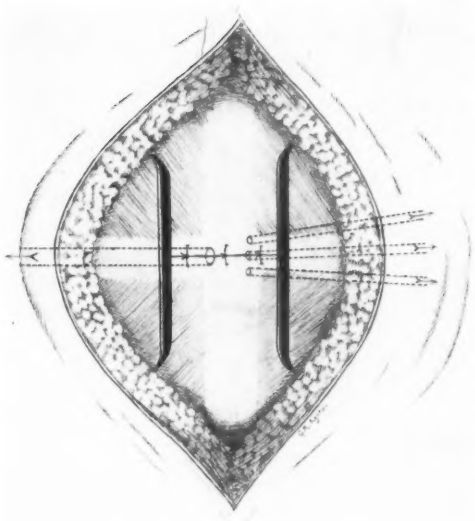

Fig. 7. Diagrammatic illustration of lines of traction exerted by umbilical ligaments, and their restoration after sutures in first aponeurotic flaps are tied.

bilical ring with connective tissue, making it relatively stronger than the upper part, a hiatus being favored at the point of lesser resistance.

The surgical principles involved in the operation for the radical cure of umbilical hernia should provide for the attachment of the umbilical ligaments at a point in the suture lines so that the pull exerted by them does not favor gaping; and should readjust the lines of traction, so that the effect of the pull will be to close rather than to open the lines of union in the reconstructed aponeurotic wall.

A transverse skin incision is made elipsing the protrusion; in the obese taking in a larger area of skin and fat, thereby reducing for cosmetics and comfort the pendulous and redundant tissue. The neck of the sac is isolated, opened, and its contents freed and returned into the abdomen; it is then amputated, and the peritoneal edges everted and approximated by a running mattress or purse-string suture. A transverse incision is made at the upper, and a parallel incision at the lower margin of the umbilical ring, thus mobilizing two lateral fascial flaps to be united by interrupted sutures. Running in the long axis of each flap is the third tendinous intersection of the rectus abdominis muscle. The edges of the upper and lower lines of incision are brought together by interrupted sutures, thus forming a double aponeurotic wall, with lines of approximation at right angles. Two figure-of-eight silk-worm-gut sutures are introduced as shown in the drawing, going deeply enough to catch the underlying fascial flap, and brought out through the skin an inch from the margin of the incision. When these are tied they relieve the strain in all directions on the aponeurotic lines of union and close the dead space in the subcutaneous fat.

On the posterior surface of the lateral aponeurotic flaps is the attachment of the umbilical ligaments. When the inner ends of these flaps are sutured together the hernial opening in the umbilical ring is closed and the attachment of the round ligament of the liver above and of the urachus and the obliterated umbilical arteries below are restored anatomically. All traction exerted by these ligaments pulls on the upper and lower ends of the line of approximation of the two lateral flaps, thus having a tendency to close and not to pull apart the sutured edges of these two flaps.

From one to three years have elapsed in six cases operated on by this technique without a recurrence.

INTRATRACHEAL ETHER ANÆSTHESIA: A NEW APPARATUS AND INTRATRACHEAL TUBE [1]

By SAMUEL ROBINSON, M.D., F.A.C.S., Rochester, Minnesota
From the Mayo Clinic

AFTER a thorough trial the intratracheal method of administering ether anæsthesia has justified its existence. There is but one contra-indication to its use; namely, the presence of a profuse purulent bronchial secretion. The method is particularly indicated in:

1. Resections of the upper or lower jaw.
2. Resections of the tongue and floor of the mouth.
3. Operations on the brain.
4. Plastic operations on the face.
5. Laryngectomies.
6. Difficult tonsillectomies requiring careful dissection.
7. Operations for cleft palate.
8. Removing tumors of the wall of the chest which involve the pleura.
9. Exploratory thoracotamies in the absence of pleural adhesions.
10. Transpleural operations on the heart and œsophagus.
11. Extensive plastic procedures for chronic empyema when there is restricted respiratory function.
12. Operations on the lung in certain rare instances.

The definite reasons for the applicability of the method in the above mentioned instances are as follows:

1. In certain operations on the head it is convenient and desirable to dispense with ether masks, cones, and inhalers which tend to soil the sterile field of operation, and since they must necessarily be removed intermittently, uneven and generally insufficient anæsthesia results.

2. To remove the anæsthetist from the immediate field permits greater accessibility, thus facilitating the work of surgeon and assistants, and diminishing the possibility of errors in the technique of asepsis.

3. The somnulent, peaceful breathing coincident with intratracheal etherization is accompanied by the minimum amount of congestion of the blood-vessels of the head; hæmostasis is thus favored.

4. The inhalation of blood, mucus, infectious material, and bits of tissue not uncommon in routine operations about the mouth and a possible source of complications, is prevented to a considerable extent by the outflowing escape of air around the intratracheal tube.

5. In certain thoracic operations already mentioned in which a free pleural cavity is to be widely opened, it is advisable, and on occasions imperative, to assist the respiratory function by insufflation of the lungs. If the intratracheal-tube is already being employed for anæsthesia the additional features of insufflation may be added promptly by certain adjustments in the apparatus.

6. The lungs of certain patients with chronic empyema are restricted in aërating surface. The necessary cramped posture on the operating table further limits the expansion of the better lung. During extensive plastic operations on such patients cyanosis is of common occurrence.

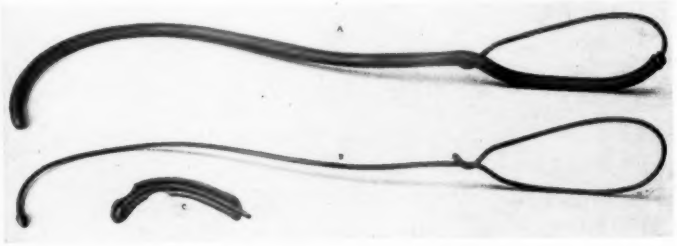

Fig. 1. An intratracheal tube. A modification of a type of urethral catheter. *B*, Olive-tipped, tempered steel stilette to guide intubation. *C*, End of tracheal tube. Hollow cup at tip for olive-stilette; two side-eyes.

[1] Submitted for publication October 21, 1915.

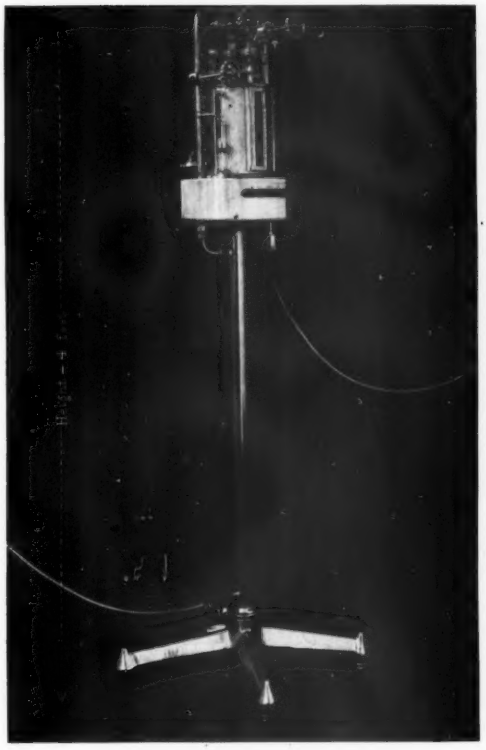

Fig. 2. An ether vaporizing apparatus for intratracheal anæsthesia.

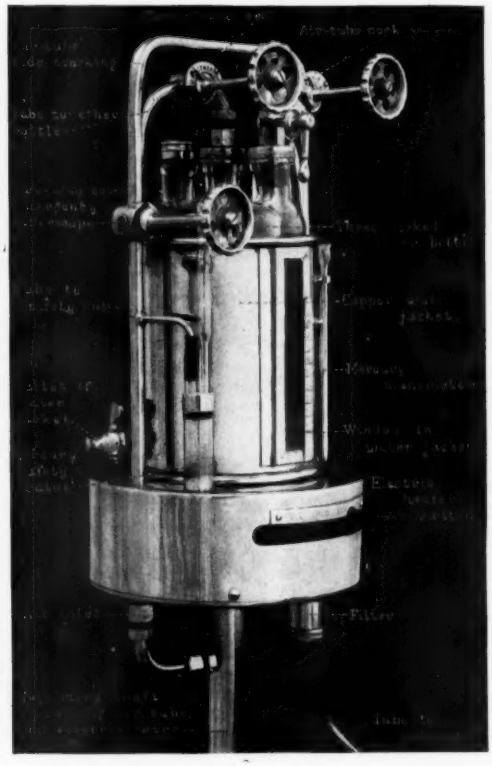

Fig. 3. Ether vaporizing apparatus showing details of upper portion.

The insufflation incident to intratracheal anæsthesia accelerates the tidal air exchange, thus relieving cyanosis.

The tube may be introduced into the trachea by any one of several methods:

1. A laryngeal speculum preferably of the Jackson type may be used to lift the epiglottis and to illuminate the vocal cords, between which the tube may be directly introduced; an excellent method requiring limited paraphernalia, slight change of posture, and moderate experience in the use of the speculum.

2. A hollow steel introducer (Cotton) of a caliber sufficient to contain the tube may be used to guide the tube across the mouth and pharynx and to point its tip in the direction of the vocal cords.

3. A woven catheter may be passed directly into the larynx by sense of touch. This procedure, owing to the absence of any guide, is difficult of execution. Trauma may result both from the introduction and from the presence of a stiff-walled tube within the trachea.

After sufficient experience with the above methods I came to these conclusions: (1) That direct vision is not essential for prompt intubation, and that the speculum is therefore superfluous; (2) that the hollow steel introducer is an insufficient guide to the tip of the tube; (3) that a tempered steel stilette within the lumen of the tube and reaching to its very tip serves as a more effective guide; (4) that an inlying soft rubber tube might cause less irritation to the tracheal membrane than a woven catheter.

Slight alterations in a certain type of stiletted soft rubber urological catheter were sufficient to provide the tube and stilette (Eynard) Fig 1. The lumen of the tube is of greater proportionate diameter to the outside diameter of the tube than is found in the usual soft rubber uretheral

catheter. There are two side eyes, provided as a safeguard against obstruction to the inflowing air and ether. The tip of the tube Fig. 1*C*, is made hollow and provides a hollow cup to retain the olive tip of the steel stilette, while the urethral catheter is generally solid at the tip, the lumen terminating in the side eye. These tracheal tubes are now manufactured in sizes from 21 to 24 F.

Technique of intubation. The stilette is bent to a curve adapted to the patient to be intubated. It is then smeared with a thin coat of vaseline and inserted into the tube, care being exercised that the olive tip of the stilette is implanted accurately in the cupped end of the tube (Fig. 1*C*). The tube is stretched gently along the shaft of the stilette and held thus by the spring grip of the stilette handle (Fig. 1A). The outside of the tube is then lubricated.

The patient lying in the flat dorsal posture is anæsthetized to the point of complete muscular relaxation. The head is raised with a two inch pillow and so placed that the face points directly upward. The intubator stands at the patients right — the assistant opposite. The jaws are well separated with a side mouth gag. The tongue seized with rubber tipped tongue forceps is drawn forward. The assistant holds the gag in his right hand — the forceps in the left. The intubator then inserts his left forefinger into the pharynx and lifts the epiglottis forward. With the right hand he gently introduces the stiletted tracheal tube across the mouth and pharynx, with care that the plane of the tube is accurately in the median line. He then causes the tip to pass behind the forefinger now supporting the epiglottis. If properly directed the tube meets obstruction at this point. The vocal cords will approximate htemselves in response to the irritating contact with the tube end. Gentle manipulation is indicated here: trauma only increases the spasm of the cords. Suddenly the tube should slip between them, at which moment the patient unless very deeply anæsthetized will cough spasmodically. This alone is ample evidence that the tube has entered the larynx. With the handle grip released the stilette is cautiously withdrawn with the right hand, the left synchronously sliding the tracheal tube over the stilette into the trachea. Under deep anæsthesia or previous laryngeal cocanization the entrance of the tube may cause no reflex spasms. Doubt may thus arise as to whether the tube is in the trachea or the œsophagus. The intubator then places the oral end of the tube against his cheek. If the tube is in proper position he will note intermittent breaths escaping from it synchronous with expiration. Meltzer refers to this as an unreliable test explaining that an œsophageal breathing also occurs and may produce the same phenomenon. Therefore he suggests that the tube be conducted well down into the throat. If it meets obstruction at a deep level it may be concluded that the trachea has been entered and that the obstructing element is the bifurcation, at which moment it is withdrawn two inches to its proper final position. If on the contrary no obstruction is met in this experiment it is presumed that the tube has entered the œsophagus. Experience nevertheless soon teaches one to gather definite and prompt conclusions as to the success of his intubation from the aggregate indications.

If the intubation has prompted spasm and coughing, it is sometimes preferable to reapply the ether mask or cone for a minute before connecting the vaporizing apparatus with the tube.

Vaporizing apparatus. There are many forms of vaporizing apparatus to provide anæsthesia when connected with an intratracheal tube. They vary in size, design, mechanism, and cost, but from the point of view of adequacy of function, they may be said to be equally efficient. Certain features in the vaporizing machines, once regarded as essential, have proved to be superfluous:

1. It was supposed that the entering air and ether mixture must be moistened by passing through water, thus supposedly imitating a function ordinarily performed by the pharynx. Apparatus devoid of such water baths have now long been used without harm and with equal efficiency.

2. The earlier machines were fitted with connections to warm the entering mixture of ether vapor. It has since been shown that it is not only quite impossible to accomplish this, but also that a mixture arising from a volume of liquid ether the temperature of which has been lowered sufficiently to cause frosting on the outside of its glass container, is yet harmless to the air passages.

3. Heat devices are yet employed in apparatus to prevent too great a lowering of the ether temperature. The object of this, however, is solely to facilitate the vaporizing of the ether and thus to economize in the amount of the liquid consumed.

4. Bacterial filters were advocated, but there is no evidence that the use of machines not thus provided has resulted in any untimely effects.

Therefore, simplicity of design and increasing lack of cumbersomeness in apparatus has resulted from six years of clinical application of this interesting method of anæsthetizing.

The apparatus illustrated in the accompanying photographs (Figs. 2 and 3) is remodeled from one previously described by the writer.[1] There are no particularly original features in its construction and yet it is presented because it has proved itself in a series of three hundred cases to be safe, useful, and reliable.

[1] Apparatus for thoracic surgery under intratracheal insufflation or positive pressure, etc.

AN OPERATION FOR THE CURE OF HERNIA BETWEEN THE ABDOMINAL RECTI[1]

By WILLIAM EDWARD FAIRFIELD, C.M., M.D., F.A.C.S., Green Bay, Wisconsin

SEVERAL years ago a woman came to me who had an enormous hernia amounting almost to complete eventration, and which had followed a central laparotomy. I operated and reoperated without success until I devised the procedure I am about to describe, and which gave me a cure. I have operated upon several patients since that time without failure in any case, and I offer my technique to the members of this society in the hope that it may meet with the approval of those who have had indifferent results from other forms of intervention.

Dr. Howard Kelly of Johns Hopkins Medical School, from whom we always expect the best and who never disappoints us, describes in his *Operative Gynecology* an operation for hernia in which he excises the fibrous ring surrounding the hernial opening, and exposes the muscles.

He then advocates suture by layers, and he includes with the sutured internal fascia the muscle fibers of the recti. I think this present technique accomplishes muscular interposition and utilizes muscle tonus much more effectually, and it further simplifies the problem of bringing like tissues into contact. When the ring of fibrous tissue is cut away and the muscles exposed it is quite a difficult matter to suture fascia to fascia, because of the tendency of muscle to bulge and interpose itself, and the further tendency of the underlying fascia to retract beneath the muscle. Again, when the fibrous ring is first excised and the cut edges of the fascia united, virtually the same condition obtains which was present when the hernia occurred (if the hernia is post-operative), and the very tissue it is essential to use in relieving the tension which leads to tearing out the stitches is thrown away.

The tough and fibrous character of the material composing the ring is admirably adapted to the anchorage of tension sutures. The method of suturing here described is not only simple, but it is also practical, being similar to the steps of an ordinary suture gastro-enterostomy.

First, let us consider the factors present and operative in the production of this form of hernia. If one will study the anatomy of the abdominal wall, it will be quickly understood that division of the fascial linea alba removes the one anchor opposing the side pull upon the recti. All of the muscles have a transverse pull upon them, and as soon as the fixed point is destroyed there is nothing to counter-balance this pull.

I have found that most post-operative herniæ of this type give a history of severe vomiting, often lasting for days after the original operation was performed. Is it any wonder that herniæ result from such effort? The binder and like devices may prevent protrusion of the abdominal contents, but they do not prevent the divergent lateral pull put upon the recti, and the result is a gradual separation, widest at the center and often drawing the muscles over almost to the crest of the ilia, leaving an unprotected space in the center, through which a ventral hernia must occur.

I believe that the practice of overlapping fasciæ in the treatment of hernia is wrong in principle, because fascial tissue will not unite to fascia when it is laid flat upon it, except where sufficient trauma has taken place to provoke a fibrous union. The tendency of these structures is to resist union and to continue to slide upon each other, as they were originally intended to do. When cure results from such a disposition of tissues it is largely an accident dependent upon trauma to the superimposed layers — if we may except the Mayo operation for umbilical hernia in which a natural physical tendency is overcome by an ingenious displacement of tissue — but success in this procedure depends little upon firm union. It is a shingling operation.

[1] Presented at the meeting of the Fox River Valley Medical Society, February, 1915.

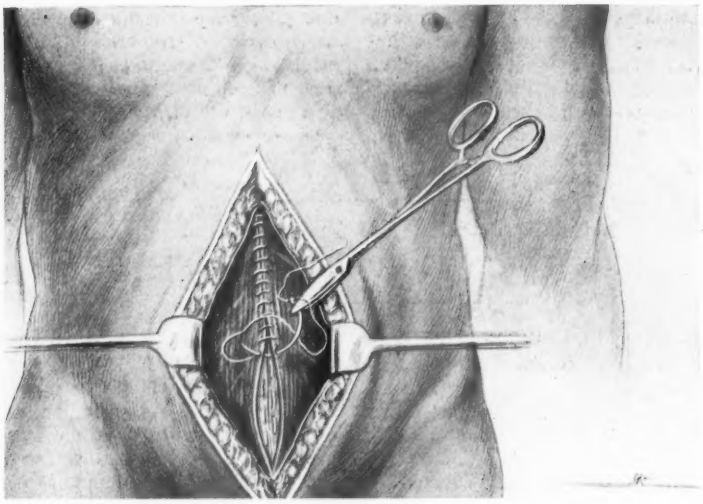

Fig. 1. Peritoneum closed. Uncut muscle sheaths being united by continuous stitch suture.

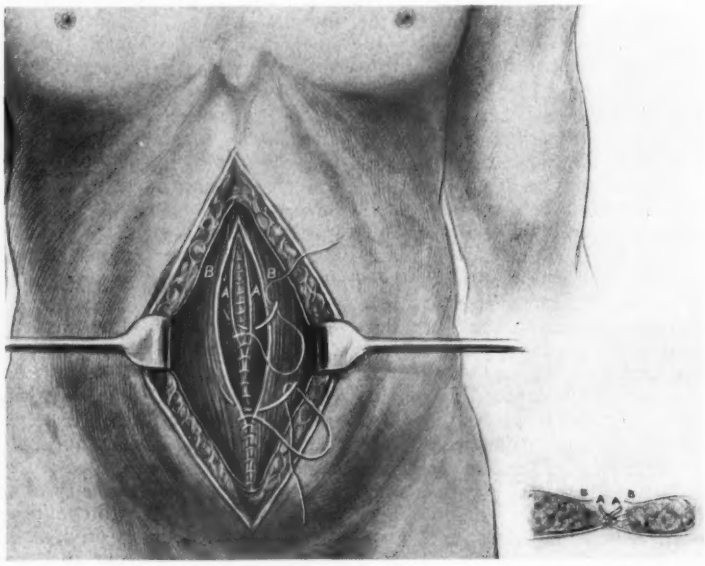

Fig. 2. Fascia split parallel to line of union and inner flaps *AA* being united by a continuous catgut suture. Outer flaps *BB* also being united. Insert does not show the second line of sutures as it should.

This operation is applicable to all forms of separation of the recti muscles, whether post-operative or not, and the procedure is the same in all cases.

The operation depends essentially upon the conversion of the two fascial spaces containing the recti into one space, obliterating thereby the natural separation of the two muscles, and in-

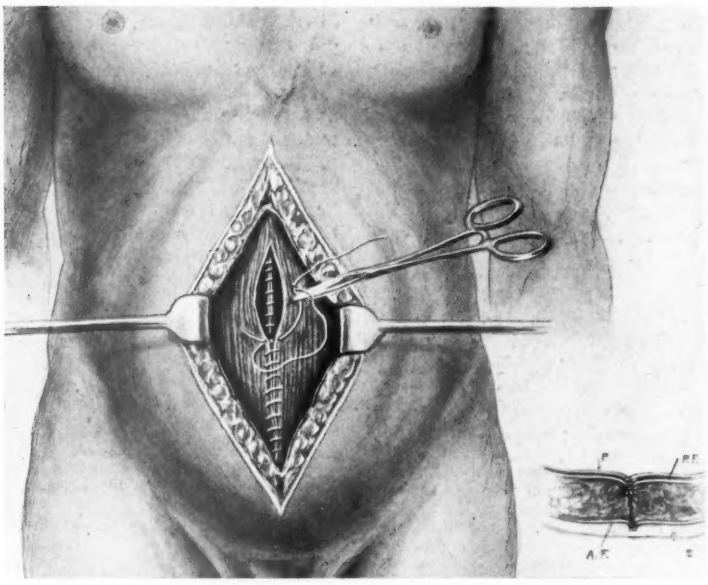

Fig. 3. Union of flaps which completes the essential steps of the operation. Tension sutures not shown.

terposing both muscles over the hernial point. An incision is carried down to the hernial sac, and a dissection is then made outward until the recti are exposed on either side. One must not become discouraged while doing this dissection. The recti will usually be found lying near the ilia at their center, but they are constantly found in their normal positions above and below the hernial opening. These muscles are enclosed in a thick fascia formed by the aponeurosis of the internal and external oblique. It is necessary to remove all the attached fat from the fascia, and for this purpose a gauze sponge is admirably adapted.

The peritoneal sac is now opened and as much of it is cut away as is necessary. On some occasions I have made an opening in the sac, and have then run a purse-string suture around the portion I judged should be removed. With the suture in place, the central portion of the peritoneum is cut away with scissors and the purse-string drawn tight and tied. When a linear incision is made it is closed with a Cushing catgut suture.

Next, the two recti are approximated by the application of a running catgut suture through the fascia covering the internal edges of the recti sheaths, much the same as we insert the first row of stitches in a gastro-enterostomy. The stitches should be four to the inch and should include the whole thickness of the fascia.

Fig. 4. Cross section showing operative scheme.

The fasciæ covering the muscles are now split the whole length of the wound, and about one-fourth of an inch external to the central union made by the running stitch. The inner fascial flaps are then united to each other by a fine running suture of catgut, just as in the second step of a gastro-enterostomy.

Next, the outer flaps are united to each other, which procedure completes the essential step of the operation; namely, the placing of both recti in one compartment. Figure-of-eight silkworm-gut sutures are made to include the skin and subcutaneous structures. The lower half of the "8" passes deeply through the recti, and acts as a tension suture. These are placed about an inch apart.

The ends are threaded upon a short section of small rubber tubing and tied. The patient is kept in bed for three weeks, and wears a binder thereafter only if she has a pendulous abdomen.

BULLET REMOVED FROM BRAIN AFTER SIX YEARS

By ERNEST MAMMEN, M.D., F.A.C.S., Bloomington, Illinois

On March 8, 1909, Z. B., age 21 years, was shot at short range by a thirty-two caliber revolver. The bullet entered the cranial cavity at the right temporal ridge about one and a half inches anterior to a line drawn perpendicular to the meatus auditorius and about one and a half inches above its level. The patient received an extensive fracture of the skull at this point, and in its track the bullet produced serious laceration of all membranes and brain tissue, penetrating the falx cerebri to the opposite side. The impact split the bullet so that one-half was found imbedded in the temporal muscle near the point of entrance. The patient was carried some sixteen miles to the hospital. There was severe hæmorrhage, shock, as well as destruction of brain tissue. Half of the bullet together with splinters of bone and loose brain tissue were carefully removed and the cavity packed with strips of gauze. The prognosis was bad.

However, there was prompt recovery with but few complicating symptoms. About the seventh day the patient escaped from the hospital under the delusion that he must get somewhere else and away from home. He was brought back and full explanation made to him, which he understood. Thereafter he remained contentedly at the hospital until his discharge. He recalls these circumstances to the present day.

About 1912 an epileptic seizure occurred in the daytime. This was followed by several others more severe than the first. I advised taking a skiagram for the purpose of locating the remaining half of the bullet. This was done by Dr. H. E. Potter, of Chicago, and the bullet found located in the left anterior fossa of the skull, within the brain tissue, somewhat below a horizontal line from the point of entrance and on the opposite side as shown by the accompanying cut. The patient was advised to have the bullet removed, but declined.

No further epileptic attacks occurred until March, 1915, when a series of them caused him to again seek my

Fig. 1. View from right side.

Fig. 2. Posterior view of location of bullet.

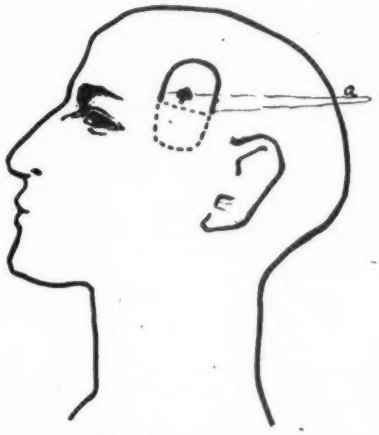

Fig. 3. Drawing showing shape of incision (*a*).

Fig. 4. Drawing of cross section showing location and shape of bullet.

advice. I again proposed skiagraphing the head for the purpose of determining whether or not the bullet had changed its position. This was done by Dr. H. W. Grote, of this city, and the bullet located in practically the same position it held three years ago. Anteroposterior skiagraphs as well as lateral stereoscopic views showed the bullet to lie about half an inch within the brain substance exactly upon the margin of the left wing of the sphenoid, or just within both the anterior and the middle lobes of the brain. This time the patient consented to have the bullet removed. The operation was done June 3, 1915 — nearly six years and three months after the accident.

The diagram illustrates the incision used. It is that of an inverted U. The lower arms beginning on a line about on a level with the location of the bullet. Small trephine openings were made at the beginning and termination of this incision and the rest of the bone opened in the usual manner with rongeur forceps. The door thus made was turned down and the dura incised. Careful exploration located the bullet exactly as shown by the skiagram. Over its location the dura had become firmly adherent to the skull. In fact at this point the membranes were all coherent and a fibrous envelope surrounded the bullet penetrating its irregularities so that in the removal there was some, though not extensive, laceration. The patient recovered promptly, after a few days of difficulty in articulating and in writing. He made incoherent sounds but was unable to produce syllables. Gradually he recovered ability to pronounce one syllable. When told to write his name or the name of his town or any other word he would write each time the same word, which was "thynd," but there was no mental impairment. There was stiffening of the muscles of the lower jaw and of the sternomastoid and its associated muscles of the neck. In three days, however, he was perfectly able to write, and his speech had improved so that he could pronounce words of two syllables. The location of the bullet was thus shown to be in the track for coördination of control of the muscles of speech and of control of the muscles employed in writing. All aphasic symptoms passed away and he was discharged on the 10th day after operation, perfectly well.

A MODIFIED McBURNEY INCISION FOR THE TREATMENT OF APPENDICITIS AND PELVIC DISEASE

By ANTHONY H. HARRIGAN, M.D., New York
Assistant Visiting Surgeon, Fordham Hospital

IT occurs not infrequently that during the performance of an appendectomy in the female, the surgeon discovers unexpectedly a pathologic condition of the uterus, fallopian tubes, or ovaries which requires operative attention. Assuredly, careful differential diagnosis, and the habitual use of bimanual pelvic examination under narcosis, should reduce this unanticipated finding to a minimum.

No technical difficulty exists if a right rectus vertical incision be used. The simple prolongation downward will afford sufficient exposure of the female pelvic organs. However, when the operator has selected the intermuscular incision of McBurney, a serious predicament arises. This incision does not give, ordinarily, sufficient room to attack complicated inflammatory lesions in the female generative organs.

In order to meet this dilemma the writer recommends the following modifications of the McBurney incision. No claim is made for originality, as the procedure is based on the ingenious operations of Weir and Fowler, which were devised to extend and to enlarge the intermuscular incision.

It is essential to recall the anatomy of the anterior rectus sheath. At this level it is formed by the union of the aponeuroses of the internal oblique and transversalis muscles, merging with the aponeurosis of the external oblique. Numerous observations on the living, confirmed by cadaver dissection, demonstrate that the aponeurosis of the external oblique fuses with the combined aponeuroses of the internal oblique and transversalis muscles at a variable point. As a rule, the line of juncture is well to the inner side of the outer border of the rectus muscle. The immediate line of contact is by loose areolar connective tissue, which is readily severed. The line of fascial adhesion is oblique, passing from above downward and inward toward the pelvic origin of the rectus muscle.

Keeping this anatomic point in mind, the operation may be described as follows:

a. After removal of the appendix through the intermuscular appendix, suture the peritoneal opening and the transversalis and the internal oblique muscles.

b. Prolong the skin incision downward and inward toward the median line.

c. Divide the aponeurosis of the external oblique to the point where it fuses strongly with the anterior rectus sheath.

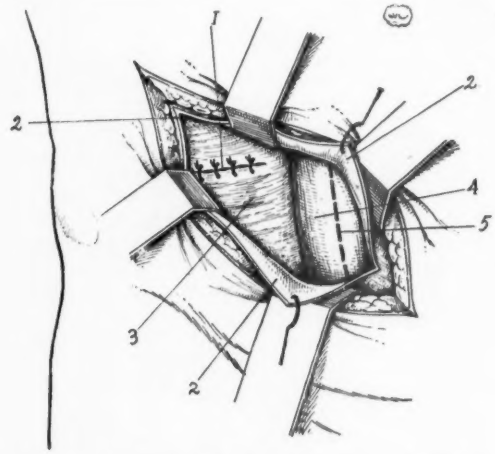

Fig. 1. (1) Internal oblique and transversalis muscles sutured; (2) external oblique aponeurosis incised to its insertion in sheath of rectus; (3) internal oblique muscle; (4) sheath of rectus; (5) incision in sheath of rectus.

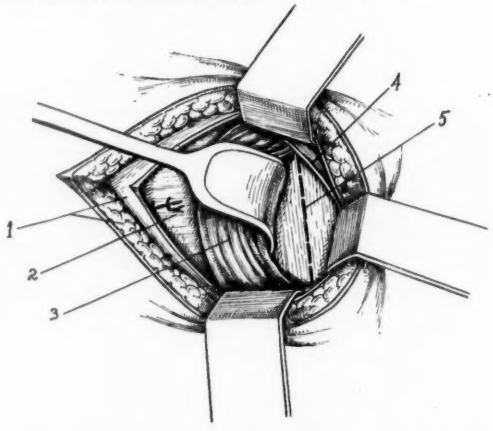

Fig. 2. (1) Skin, fat, and external oblique muscle; (2) internal oblique muscle; (3) rectus retracted outward; (4) cut edge of rectus sheath retracted; (5) incision through peritoneum.

d. Incise the anterior rectus sheath parallel to the line of fusion of the aponeurosis of the external oblique muscle, leaving a sufficient margin internally to suture.

e. Free, displace, and retract outward the rectus muscle.

f. Incise the peritoneum.

g. The closure is simple. Following the suture of the peritoneum the rectus muscle is replaced, and the sheath sutured. The external oblique muscle is sutured with a continuous catgut suture and the skin margins approximated in the usual fashion.

The advantages are the employment of one incision for the combined operation, which is desirable when the patient has not been informed of the likelihood of a second operation. No muscles or nerves are divided; complete anatomic restoration of the abdominal wall is obtained. A possible objection is an unduly long skin incision. This can be materially shortened by shifting and sliding the skin incision downward and maintaining the displacement with retraction. The writer has had no difficulty in removing adherent and fixed inflammatory tubal and ovarian masses through this incision.

THE USE AND ABUSE OF LANE PLATES

By JACOB FRANK, M.D., F.A.C.S., Chicago

THE introduction of Lane plates in the operative treatment of fractures was a notable addition to our armament in caring for such conditions. The immediate acceptance by the surgical profession of this method was due in part to the distinction made between functional and anatomical results secured by the hitherto routine non-operative treatment of fractures, a distinction brought in the foreground by the marked development of röntgenology and its application to this branch of surgery. We all know that a good functional result is not always entirely dependent upon a good anatomical result, as shown by the X-rays; nor is the reverse true. Nevertheless, the lay mind, influenced by the visions of proceeds resulting from malpractice suits, often confuses the two and thinks they are interdependent, in which opinion he is supported only too frequently by members of the legal profession. The desire to secure good results, both functional and anatomical, therefore, was the immediate *vis a tergo* behind Lane's method in its adoption as a standard method of the treatment of fractures.

The gradual advancement in our aseptic surgical technique has widened the scope for the operative treatment of fractures, so that one school — that of Lane — even advocates the open reduction of all simple fractures. It is only fair to state that many are of the opinion that operative treatment should be applied only in the exceptional case. Nevertheless, Lane's views have been adopted by the profession the world over. The application of this method by surgeons less skilled than the originator and less cognizant of the extreme asepsis required for good results, has resulted in incalculable harm. It has fallen to my lot to get many patients previously operated upon, and presenting indifferent and bad results. I realize that patients with good results would not return; and yet, is the method as now used indiscriminately by anybody and everybody professing to be a surgeon, worth while? This doubt has led me to look up the literature on the matter.

The necessity for absolutely perfect asepsis in bone surgery is recognized as axiomatic, and yet such asepsis is not synonymous with a good result. Sheen (1) says the screws often loosen in rigidly clean cases, thus making the plating method in these cases, as far as fixation of fragments is concerned, inefficient. Hey-Groves, before the International Medical Congress (1913), stated the same thing. A view on this point from Lane's clinic is enlightening. Sampson (2) writes concerning Lane's cases that the rarefying osteitis at the basis of the loosened screws *probably*[1] does not occur without the introduction of organisms. It is to be noted that Lane himself is not certain that absolute asepsis guarantees a good result.

Needless to say, the metallic bodies binding the fragments do not form a scaffolding for the new bony growth. This is amply demonstrated by the persistent sinuses which heal only after the plate has been removed. Often enough, absorption and disintegration of the bone surrounding the plates and screws occurs. Edington (3) reports cases in which some sinuses healed after removal

[1] Italics, my own.

of plates. Others refused to heal until a tertiary operation was performed, at which the carious bone around the nail-holes was curetted. Estes (4) states that the metallic appliances produce so much "irritation" long after bony union has occurred that they must be removed. It may not be amiss to mention here, as illustrative of the irritating influences of foreign bodies on bone, Ewalds (5) experiences with the Steinmann nail-extension method. Unfortunate sequelæ followed in 7 of 29 cases. The fractures of the leg, undisturbed themselves, healed ideally, but the holes left by the nails were the seat of recurring inflammation, so that healing did not take place until two and one-half years after operation.

As for stimulating the osteogenetic power of the fragments, the bone plates do just the opposite; they retard osteogenesis (Martin and Barton, 6). Sheen (1) states that the firm fixation by the plates and screws does not favor callus formation. This is especially adverse in operations on old ununited and malunited fractures, where the normal reparative power of bone is already considerably lessened. In addition, the bone along the screw-tracts or nail-tracts is resolved, so that fracture may occur at this site (Lilienthal, 7).

Following plating operations, stiffness in the neighboring joints frequently occurs. This is almost constant in adults in the knee-joint, after plating of the femur (Sheen, 1). Sampson, writing from Lane's clinic, makes the same statement concerning cases in children. To return to Ewald's work with the nail-extension method: In 6 cases, out of 29, the nails had been driven too close to the ankle (Steinmann states they should be at least two and one-half cm. away from joint), so that painful ankylosis of all joints in the ankle and foot resulted, with severe atrophy of the bones in one case. In others, spur formation on the calcaneum seemed to have been excited, resulting in extremely painful heels.

Upon superficial thought, it might appear that a fracture operated upon would heal quicker, thus permitting the patient to return to his employment sooner than if non-operative treatment had been pursued. This is not so, as the same fixation with splint or cast for an identical length of time is required as with conservative treatment. Lane gives forth the dictum that "no curtailment of the period of fixation is permitted."

Perusal of the literature teaches one that—

1. Plates do not always fixate the fragments, as the screws often enough are loosened by an aseptic rarefying osteitis.

2. Plates do not act as a trestle-work; on the contrary, they cause destruction of adjoining osseous tissue.

3. Plates do not stimulate osteogenesis; on the contrary, the primary firm fixation is a distinct hindrance to new bony growth.

4. Unfortunate sequelæ resulting from bone plating, such as persistent sinuses and stiffness in neighboring joints, are not at all infrequent.

5. From an economic point of view, no time is gained for the patient by operative treatment.

From a large and varied experience, which thirty-three years of active practice has given him, during which time he has seen the gradual shift from the non-operative to the operative treatment of fractures, the writer would enter vigorous protest against this wholesale change. The art of setting fractures by non-operative means is fast becoming a lost one. Fractures were sustained, and the same reduced, long before the introduction of the röntgen rays and operative methods, and useful limbs obtained. This does not imply that we should not make use of the röntgen rays. By all means let us use it, but we should not overestimate the value of the X-ray plate. In other words, our goal should be a good functional limb and not necessarily one that is restored to anatomical perfection. Such, in the vast majority of cases, can be secured with the old-fashioned methods used before the introduction of the operative treatment; and in observing this conservatism we would merely be applying the "Golden Rule" to this branch of surgery; for surely, if the reader or writer sustained a fracture he would first permit Nature to try her hand at the healing process before calling upon surgical intervention.

Many instances might be cited which, to my mind, demonstrate the folly of hurried interference in an operative way; likewise, many cases which clearly show Nature's reparative powers with very little of the surgeon's aid beyond that of reduction and putting the parts at rest. I will limit myself to the detailed report of two cases, illustrative both of the possibilities for evil inherent in bone plates, and the value of conservatism even under unfavorable circumstances.

Henry H., age 21, Russian. October 21, 1914, fell off a slowly moving wagon when wheel slipped off axle. Sustained a compound fracture of both bones of right leg. Was brought to Cook County Hospital where, three days later, he was operated upon, the fragments being replaced in position and the wound sutured. However, good position was not maintained in a fracture box, so one week later Lane plate was inserted to hold the fragments together. At the same time a large denuded area in the operative

field was covered by a whole skin graft. Cast applied. The skin graft failed completely. The leg became greatly swollen, remaining so for one month, when it was decided to remove plate. After this was done, the cast was reapplied, remaining in place ten weeks, when a fracture-box was substituted. There was only slight bony union and a large raw area containing four or five sinuses leading down to bone. Amputation of limb was considered, when patient took matters into his own hands and had himself removed to the German Hospital under my care last February. Under local anæsthesia, the sinuses were probed and curetted, several pieces of dead bone (sequestra) being removed. Edges of greatly thickened involucrum were freshened, as well as the skin edges and granulating surface. Sinuses packed, wound kept clean and posterior splint involving whole leg and foot applied. Filling in of the sinuses slowly but surely took place, new skin gradually crept in from the edges, and he now has a fairly good functional limb, being able to get around very nicely with the aid of a cane.

Frank H., age 39, German. June 16, 1914, was riding on a motorcycle when hub of heavy truck brushed against his left shin bone, throwing him on to street. He sustained a fracture of both bones of the leg, compounded by a small opening in the skin through which a tibial fragment had torn its way. He was taken to the Columbus Hospital, where leg lay between sand bags for five days, the external wound being dressed sterilely in the meantime. Then plate was applied and plaster cast put on, a window being cut into cast for purpose of dressing wound, as drainage was used at operation. Five weeks later (August 1), plate was removed and ambulatory splint replaced the cast, this splint remaining on about eight weeks. All this time the wound was being dressed. Complete healing finally took place in November, five months after his original operation. The patient used crutches until December 20, when he was able to get about with the aid of two canes. He discarded one cane at the end of January, 1915, but two months later (March), he was forced to renew use of both canes on account of increasing pain in leg. The pain soon became of such severity as to incapacitate him entirely when, March 8, he came under my care at the German Hospital.

At this time, there was a marked anteroposterior angular deformity of left tibia, the integument over which was considerably reddened and excruciatingly tender. No rise in temperature accompanied this process. The tibia seemed to be on the verge of bursting through the skin. March 13, he was operated upon, two incisions being made, one over fibula, the other over the internal aspect of tibia. Both tibia and fibula were rebroken in line of old fractures, excess callus chiseled away, some sequestra removed, and leg straightened. The wound over the fibula was closed tightly, but the internal incision was drained with gauze down to the bone to take care of oozing. Ambulatory splint applied while patient was still on operating table. However, the fragments tended to sag, so after twenty-four hours a posterior plaster cast splint, molded previous to operation, was applied. The gauze drain was removed in forty-eight hours. There was primary union of wounds. The cast-splint remained on for four weeks, when a cast involving only the leg and foot (not the thigh) was applied. This remained on for three weeks, when a small leather posterior splint was substituted. He now has a good functional leg, being able to get about without help of any kind. The bone is thoroughly united.

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CORRESPONDENCE

Nephrectomy During Pregnancy

To the Editor: In the June 1915 number of Surgery, Gynecology and Obstetrics, Dr. Anthony H. Harrigan reports a case of "Nephrectomy During Pregnancy," and gives summaries of cases previously reported. I wish to put on record a case which occurred in my private service at the Macon Hospital. The case presented no especially unusual features so I will report it briefly:

Mrs. A. R. T., age 20, was admitted to the Macon Hospital on December 23, 1913, being then five and one-half months pregnant. She gave a history of having been perfectly well until four days before admission when she was seized with a chill and intense pain in the region of right kidney. The pain had been continuous since first onset, though varying in severity. She has had several chills and her temperature varied from normal to 103°.

Examination shows a small, ill-nourished woman, weighing ninety pounds. Temperature on admission, 102°. Marked tenderness present in region of right kidney, pain being severe enough to require repeated injections of morphine.

Urinalysis. Urine highly colored; specific gravity, 1028, with a large trace of albumin and a large number of granular casts, red blood-cells, and pus-cells.

The white blood count on admission was 32,000. Twelve hours after admission the temperature went to 103.3° at which time I operated.

Operation. Mayo incision, exposing kidney. Kidney delivered and showed three or four large yellow nodules with a number of small ones. The condition of the patient was very poor, the pedicle was clamped and the kidney rapidly removed. The patient was put to bed, with Murphy drip, tap water. Recovery was normal, the temperature going up to 105°, six hours after operation, and reaching normal in 24 hours. Twenty-four hours after operation the patient's face became slightly swollen. During the first 24 hours 290 ccm. of urine was obtained; second day, 410; third day, 1200; fourth day, 1910; fifth day, 2200; sixth day, 2480. Later history: On February 7, 1914, with normal urinalysis and for no assignable reason she went into labor and was delivered of a seven months child which lived a few minutes. Since this time she has been perfectly well.

Diagnosis: Septic infarcts of the kidney.

In this case I thought there was a better chance for the patient to recover if nephrectomy were done than if nephrotomy were done. In 3 cases in the past two years I have done nephrectomy twice in ill-nourished women with surgical kidneys, and nephrotomy in one instance in a strong woman three weeks post-partum. All three cases made uneventual recoveries.

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BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

The Practical Medicine Series. Vol. V., edited by Isaac A. Abt, M.D.; Orthopedic Surgery, edited by John Ridlon, M.D., and Charles A. Parker, M.D., Chicago: The Year Book Publishers, 1915.

Principles of Human Physiology. By Ernest H. Starling, M.D., Lond.; F.R.C.P.; F.R.C.S.; Hon. M.D., Breslau; Hon. Sc. D., Cambridge and Dublin. Second edition. Philadelphia and New York: Lea & Febiger, 1915.

The Book of the Fly. By G. Hurlstone Hardy. New York: Rebman Company, 1915.

Student's Textbook of Hygiene. W. James Wilson, M.D., D.Sc. New York: Rebman Company, 1915.

The Clinics of John B. Murphy, M.D., at Mercy Hospital, Chicago. Vol. IV, Nos. 4 and 5. Philadelphia and London: W. B. Saunders Company, 1915.

Diseases of the Nervous System. A Textbook of Neurology and Psychiatry. By Smith Ely Jelliffe, M.D., Ph.D., and William A. White, M.D. Philadelphia and New York: Lea & Febiger, 1915.

A Textbook of Histology. By Rudolph Krause. New York: Rebman Company, 1915.

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The Medical Clinics of Chicago. Vol. I, No. 2. Philadelphia and London: W. B. Saunders Company, 1915.

Emergency Surgery. By John W. Sluss, A.M., M.D. Third edition, revised and enlarged. Philadelphia: P. Blakiston's Son & Co., 1915.

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Oral Surgery. A Treatise on the Diseases, Injuries, and Malformations of the Mouth and Associated parts. By Truman W. Brophy, M.D., D.D.S., LL.D., Sc.D., F.A.C.S. Philadelphia: P. Blakiston's Son & Co., 1915.

Orthopedic Surgery. By Edward H. Bradford, M.D., and Robert W. Lovett, M.D. Fifth Edition. New York: William Wood and Company, 1915.

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